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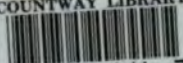
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**THE PERITONEUM**  
**VOL. II**





# THE PERITONEUM

VOL. II

DISEASES AND THEIR TREATMENT

BY

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**St. Louis**

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# THE PERITONEUM

## VOL. II

### PART I

#### CHAPTER X

#### CLASSIFICATION OF PERITONITIS

Any disease which is an expression of end result or complication of other disease must necessarily present unusual problems in classification. Peritonitis, usually being the result of secondary invasion into the peritoneal cavity of an infection arising from some other organ, partakes somewhat of the characteristics of the disease of the organ from which the infection is derived. These differences have to do with the suddenness with which the invasion takes place, the location of the organ with relation to the peritoneal cavity, and, finally, the character of the organisms it is prone to harbor. Notwithstanding the protean character of the primary affections that may give rise to peritonitis, by keeping in mind the topography of the infection and rate of onset, together with the individual character of the disease of the organ primarily at fault, a classification of the peritonitis is possible which is adequate to the needs of the surgeon. The pathologist with his own too limited knowledge of the disease in all its phases should not be too free to find fault. If it were possible to do so, it would be desirable to classify the disease according to the offending organism. This is in fact possible in a few instances, as in the case of pneumococcic, gonococcic and tuberculous peritonitis. In most instances, however, the determination of the causative organism clinically is

usually not possible because of the multiplicity of the bacteria present. In such instances the classification must be based on site of origin, the method of invasion, and the topography of the disease. Because of these interrelated factors it is much easier to analyze the various forces at play in a concrete case at the bedside than it is to separate them out for academic discussion.

From the foregoing it is evident that three basal factors for classification, neither of which can be neglected in a clinical study, present themselves for consideration. These are the area involved, the organ from which the infection spreads, and finally the type of bacteria playing the dominant role.

**The Area Involved.**—In the first, the extent of the disease may be made the basis of classification. The importance of the extent of the disease to the clinician lies in the fact that, other things being equal, the severity of the disease in a general way runs parallel with the area involved. The difficulty here lies in that the disease is a process and not a state. Because of this, a given patient may belong in one group in one period of the disease and in quite another at a different or terminal stage. These difficulties are increased enormously by the fact that it is impossible to determine with exactness clinically in just what anatomic stage the disease may be at any period of the observation. However, by taking into account the organ from which the infection arises, the manner of onset and rate of progress, surgeons are able in a measure at least to prognosticate the future course of the disease. In this way certain inflammations, it can be determined, will remain local, as in gonorrheal perisalpingitis, others again as in periappendicitis, remain localized in the majority of cases or can be made to do so by timely action. Others again, as in perforations of an ulcer, will most certainly spread unless hindered by the most energetic measures. We may divide the inflammations, therefore, into localized, spreading and diffuse.

**Localized Peritonitis.**—A localized peritonitis in its strict sense is one which has no tendency to spread beyond the tissues actually involved, just as fibroma has not. In these cases there is usually no complete solution of continuity of the organ involved, the peritoneum being set into a state of reaction by the dissemination of the toxins of the infective process, and not by the diffusion of the

bacteria themselves. The most familiar example of this type is seen in appendicitis when the wall of this organ is inflamed, producing a periappendicitis, but in which there is no perforation of its wall. If organisms do escape from the hollow of the organ, this event is anticipated by the formation of adhesions with neighboring structures and as a result of this precautionary reaction, a spreading infection is avoided and the most serious result is the formation of a localized abscess.

**The Spreading Type.**—The spreading type while advancing does so against resistance. The organism places barriers before its advancement which the disease can not fully overcome. Here bacteria escape and by their multiplication gradually extend the process over more and more of the surface of the peritoneum. The extent of the spreading is then dependent on the relative virulence of the organisms and the degree of resistance of the tissues. For instance, in certain types of peritonitis following appendicitis the infection begins at the site of the appendix, becomes partially walled off, hesitates for some hours or days, then spreads toward the diaphragm or into and across the pelvis. The experienced clinician can follow this progress in his mind's eye as accurately as he can the progress of an erysipelatous lesion of the skin.

As opposed to these types, either because of slight virulence of the invading organism or because of the slowness of invasion, are those cases in which the surrounding peritoneum walls off the invading host at some stage of the progress, and thus protects the surrounding peritoneum from further invasion. These become then secondarily localized processes. The seriousness of this type is dependent less on the extent of the disease than on the character of the walling-off process. An abscess involving an area greater in extent than either a diffuse or spreading peritonitis may be quite innocent of harm because, being completely walled off by adhesions, it is essentially extraperitoneal and the surgeon at operation is dealing really with an abscess communicating with the surface. For instance, I observed not long ago one child, with a huge abscess taking in one-third of the abdominal cavity, recover promptly after drainage, and another which died following an infection of a much smaller area. The former required three weeks for its development, while the latter

ran its course in three days. On the other hand a spreading peritonitis which has become localized may break through its walls and become diffuse.

**The Diffuse Type.**—The diffuse type may be likened to the most malignant sarcomata, against the spread of which the organism is utterly helpless. The helplessness of the organism may be due to the variety of the bacterium liberated into the peritoneal cavity, to the coexistence of foreign substances, as fecal masses, with the infecting organisms, to the presence of digestive ferments which make adhesion formation impossible, or, finally, to the general state of the individual, there being, as we say, a lessened constitutional resistance, the meaning of which we sometimes know and sometimes we do not.

A division of the disease according to the area involved is not scientific, yet it is very useful in practice. The expert surgeon learns to judge these factors, just as the trained oncologist in viewing a tumor is able to say what its subsequent course will be. In both instances it is often quite as much intuition as science that leads to skill along these lines. A peritonitis involving a very small area may be essentially diffuse because the organism will not at any time be able to cope with it. This may find expression in the general appearance of the patient. Thus a perforated duodenal ulcer or a perforation of a typhoid ulcer gives rise to a peritonitis which is essentially a diffuse peritonitis from the beginning, for a knowledge of the pathogenesis indicates, and clinical experience has proved, that such lesions will continue to spread. A spreading peritonitis of greater area than those above noted may be less serious to the patient because the peritoneum is obstructing in part its advance by the formation of adhesions. This ability to cope with the disease on the part of the patient may be read more in the general bodily reaction than in the physical examination of the abdominal region.

In considering the above classification, therefore, the surgeon must have in mind not extent but tendency. This tendency is expressed by certain pathologic processes already considered in the chapter on general pathology (Vol. I), but which are not subject to inspection in the unopened abdomen and the observer must base his deductions on the probable source of the infection, and



the variety of the organism causing the infection, and the general reaction of the patient. The above classification alone, therefore, is not sufficient for our clinical requirements.

**The Organ from Which the Infection Originates.**—The second basis for classification is in a measure corollary to the preceding, but since it frequently forms the basis of the determination of the former it requires a separate consideration. It has to do with the organ from which the infection is derived. The importance of such a determination has already been indicated. Perforations of duodenal or typhoid ulcerations it was noted were followed by dire consequences, while in other organs like lesions might be much less so. The first problem for the surgeon, therefore, when confronted by a patient exhibiting the signs of peritonitis is to determine the organ at fault. This is so because this problem must often be considered before the elemental diagnosis of peritoneal involvement can be made. Thus it may be between gallstone colic and perforated duodenal ulcer that a diagnosis must be made. That the importance of the determination of the organ from which the infection has arisen is the elementary factor is abundantly testified by the fact that this nomenclature dominates the field. We speak of perforated duodenal ulcer, but think of the inevitable spreading peritonitis.

Certain qualifying adjectives must be appended to the organ from which the infection is derived in certain cases. Thus in chronic ulcer a thickening of the gut wall is coincident with adhesions to the surrounding peritoneum. We may therefore speak of a perigastritis, meaning a slowly developing peritonitis, or if a collection of pus forms, a perigastric abscess, meaning a localized suppurative peritonitis. A gall-bladder lesion may give rise to a surrounding peritonitis which we call a pericholecystitis. It is only after the inflammation extends beyond the organ that we resort to the general term peritonitis.

So important is the determination of the organ from which the peritonitis springs that this classification will be made the basis for discussion, for upon it depends the determination of that more important factor, the clinical course. It is not important to detail the various groups, for such division is entirely arbitrary and to be complete must needs be coextensive with the possible sources

of peritoneal infection. An attempt at completeness need not be made since each surgeon constructs his own. The more common types, most readily recognized clinically, will be studied as entities.

The mention of appendiceal, perforating, and gonococcic will call to mind the less frequent forms.

**The Specific Causative Organism.**—Finally, the actual agent producing the infection may be made the basis of classification. Though a bacterial classification might at first thought seem the most scientific, it is not so. The reason for this is that the capacity for harm of any organism is much influenced by the associated conditions. As was noted in the chapter on general pathology the addition of foreign bodies to infected material enormously enhances the infectivity. The addition of excreta or secretions or ferments inhibits the walling-off process and in this way allows free play to the virulence of the organism. The chief difficulty, however, lies in the fact that in most cases of peritonitis there is a multiplicity of organisms. This must always be true of perforative cases. It is only when a peritonitis results from an extension of a specific process elsewhere that a single organism is at play.

The limitation of a classification based on the specific organism is due therefore to the factors that surround an ethnologic classification of the American people. The vast range of possibilities is due not only to the great variety, but quite as much to the conditions under which they develop. Two infinite variables interacting present possibilities scarcely calculated to encourage hopes for classification.

These remarks are not calculated, however, to discourage a study of dominant types. The common organisms are the streptococcus, staphylococcus, colon bacillus and less commonly the pneumococcus, gonococcus and pyocyanus. Even when a large number of other bacteria are associated, the dominant type may still disclose itself with sufficient clearness to enable the surgeon to determine his course of action. For this reason a determination of the flora in every case is of educative value to the surgeon. The mere recognition of a condition as peritonitis, like carcinoma, is of value, yet in both conditions extensive analytic studies broaden the conception wonderfully.

In those instances in which a single organism is at play, a bacterial diagnosis assumes a more dignified, even dominant role in nomenclature, as in tuberculous peritonitis, less so in gonococcal and pneumonic peritonitis. In the case of the latter organism a clinical diagnosis is possible with a considerable degree of accuracy. In the cases of acute perforative peritonitis a preoperative determination of the organism at fault is purely conjectural, and the certain determination of the variety of organisms is a task for the trained bacteriologist to work out from material obtained at operation. Even with such material the problems have been unsatisfactorily worked out as will be noted in the discussion of the etiology. Surgeons have not been insistent on a more exact knowledge because, save possibly in the case of pyocyanus, their course of after-treatment is but little influenced by the findings of their laboratory confreres.

According to such a scheme, in every case of peritonitis the surgeon must place the disease in one of the categories of each of the following groups:

As to extent: 1. Localized. 2. Spreading. 3. Diffuse.

As to organogenesis: 1. Appendiceal. 2. Cholecystitic. 3. Gastrointestinal perforations. 4. Genitourinary. 5. Metastatic and Thrombotic, etc.

As to causative organisms: 1. Streptococcus. 2. Colon Bacillus. 3. Staphylococcus. 4. Pneumococcus. 5. Gonococcus, etc.

A complete classification in a concrete case requires a consideration of all these factors. In a given case, for instance a diseased appendix, the organ indicates its source, the course of the disease indicates whether or not it is spreading. Because of the organ at fault it is probable that the colon bacillus is the dominant organism, a problem capable of final solution only after the abdomen is opened. It is only when the surgeon has habituated himself into the making of such a classification in every instance that he obtains that breadth of view necessary to the most efficient handling of the therapeutic problem.

The foregoing is an outline of the essentials in the classification of peritonitis. This will be augmented in individual cases by topographic or pathologic detail to suit the observer. A new classifi-

cation of peritonitis was to the early abdominal surgeon what the invention of a new speculum was to the early gynecologists, each invented his own and it was for him the best. Now, however, since the more fundamental factors have become common property a more simple classification is ample, leaving it to the knowledge of the individual to vary the detail just as the nurse constructs for each patient a temperature chart all his own.

## CHAPTER XI

### ETIOLOGY OF PERITONITIS

Generally speaking, the genesis of peritonitis is dependent upon the advent of bacteria in the peritoneal cavity. The method of entrance and variety of organism is subject to such great variation that the etiology of peritonitis is one of the most complex problems in clinical medicine. The variety of organisms which gains admittance into the peritoneal cavity is of considerable importance in etiology, but the variation in virulence of any given species is of equal importance. The method by which the bacteria gain entrance is of even more importance than the variety of organisms. These factors can best be studied in the discussion of the various subdivisions of peritonitis which surgeons usually employ. While bacteria constitute the chief, they are not the sole, cause of peritonitis. Chemical substances may produce an inflammatory reaction, which, while seldom leading to the death of the patient, often is followed by lasting minor disabilities.

**Chemical Peritonitis.**—Chemical peritonitis has been the product of laboratory experimentation, though in a few instances it has a clinical significance. Croton oil, cantharides, iodine, and turpentine have been the substances usually employed for the purpose of the experimental production of this form. Pawlowsky found that two and one-half drops of croton oil produced a fatal hemorrhagic peritonitis in rabbits. The same author found that quantities as small as 0.1 gm. trypsin produced a fatal hemorrhagic peritonitis. When any of the substances above mentioned are brought into contact with the peritoneum, a hyperemia ensues, resulting in a great widening of the service vessels and the assumption of function by the potential vessels. If the action is more intense, an exudation results both of leucocytes and of serum. These processes have already been sufficiently described in the general chapter on inflammation. In the use of such substances it may be an error to speak of the fatality as be-

ing due to the peritonitis, possibly the peritonitis is but a concomitant factor, death being due to the toxicity of the drug employed. I am led to make this remark because in my own studies I was amazed to find that a grain or two of magnesium sulphate injected into the peritoneal cavity of a rabbit proved quickly fatal without there being any observable change in the peritoneum itself.

The chief interest in this variety of peritonitis is that any chemical substance introduced into the peritoneal cavity which acts as an irritant invites the growth of bacteria. This is brought about by the exudate which results from the irritation. The same is true of physical agents. It has repeatedly been proved that after the peritoneum has been irritated, infections become established which in the unirritated peritoneum remained innocuous. Since the use of chemicals in the peritoneal cavity has been discontinued, this influence is but little encountered. To appreciate the importance of this question, one must read the literature of the early antiseptic period.

The escape of fluids from hollow organs may cause a peritonitis. Often these are contaminated with bacteria and because of these a progressive peritonitis is produced. Ruptures of the urinary and biliary bladders are familiar examples of this. Exudates from strangulated tissue, as the omentum, cysts or tumors may produce marked reactions. In each of these instances the tissue reaction overcomes the irritation unless the absorption of toxins produces associated conditions.

The bursting of colloidal and pseudomucinous cysts is the common physical and chemical cause of chronic reaction on the part of the peritoneum. This variety will receive attention in a separate chapter. The bursting of other aseptic cysts, e.g., echinococcus, may give rise to more or less irritation, as may the bursting of blood and lymph cysts which have undergone certain chemical changes before they rupture. The contents of blood and lymph cysts which rupture without previous infection are absorbed without reaction.

In certain instances a chronic reaction may arise from unknown causes. These cases are collected in a separate section corollary to the chapter on tuberculous peritonitis. Localized productive reactions may result from inflammatory reactions in the neigh-

borhood of the peritoneum, themselves infective in character, but in which the infectious material does not reach the peritoneal cavity. This is seen most frequently in juxtaposition to ulcerations of the digestive and biliary tracts.

**Bacterial Peritonitis.**—The very vast majority of peritonitides are the result of the invasion of the peritoneal cavity by bacteria. To such an extent is this true that when the term “peritonitis” is used without qualification this variety is meant.

Only with the development of modern bacteriology was an adequate discussion of peritonitis possible. Soon after this development the problem of peritonitis received the active attention of a host of competent bacteriologists. As a matter of fact but little has been added since the active campaign of the period ten or twenty years subsequent to the discoveries of Koch.

As in any infection, the development of peritonitis depends upon the kind and virulence of the organisms and the constitutional and local resistance of the individual. The local and general resistance to bacteria has been abundantly studied, and we possess records of many researches calculated to clear up the problem. The fact remains that occasionally a postoperative peritonitis appears even in the hands of the most expert, the cause of which can not be traced. That bacteria are rapidly absorbed from the peritoneal cavity is well known. After they reach the blood stream they no doubt encounter the same resistance as when injected directly into the blood stream. Werigo studied this phase. He found that bacteria so introduced soon found their way to the liver, spleen, and lungs. Here, according to him, they are taken up by the phagocytes. The length of time that elapses before this takes place has been the subject of a varying interpretation. Bail injected the bacteria into the pleural cavity and, instead of studying them in the section as Werigo did, recovered them from the tissues by cultural methods.

Recently Buxton studied the fate of bacteria introduced into the peritoneal cavity. He employed typhoid bacilli, removing fluid from the peritoneal cavity as well as plating tissues removed from the various organs. He concludes that the body fluids, unaided by the cells, have the power of destroying bacteria. Following this there is a slower destruction of the bacteria due to the



action of phagocytes on the surface of the peritoneum. Bail and Buxton agree that the destructive action of the serum is less after the cells appear. This loss Hoke attributes to the absorption of complement by the cells. Even in the organs Buxton concludes that there is an active destruction by the serum at first and later the phagocytes become active. The results above noted are not constant. The disappearance from the peritoneum soon after introduction does not prove that they have been destroyed because inanimate bodies may suffer a like fate. Moreover the numbers of bacteria are so great that an attempt at securing results by plating must be subject to great error.

My own studies lead me to believe that relatively few of the bacteria reach the blood or lymph stream but that the larger proportion is destroyed by the serum. When the peritoneal fluid is abnormally increased by previous irritation by a foreign body or a chemical, this bactericidal power is lost and instead of the virulence being decreased, it may actually be increased. Bacteria placed into a peritoneal cavity for 30 minutes and then cultured are less toxic to animals than the culture which has not been so placed. In order to determine the fate of bacteria it is necessary to section peritoneal tissue. By this means large clumps of bacteria surrounded by fibrin and cells can be demonstrated.

Obviously when the subject is considered under clinical conditions results are bound to be at variance. Here the individuality of the organism as well as the patient is subject to the greatest variation. In laboratory experimentation one can go back to the original culture for comparison of virulence but when a clinical problem arises either the source is unknown or is not available for comparison. The problem is still more complicated because substances other than the bacteria are usually introduced at the same time that the organisms gain entrance, and the trauma of the operation either acts as or produces a foreign body which may prove a continued source of irritation.

My own researches along this line, which were instituted for the purpose of interpretation of the results of a study of pure cultures under clinical conditions, failed to secure uniform results. It seems to me, however, that the associated conditions such as trauma from the operation or foreign bodies left, such as ligatures or dead

tissue, are as important as the strain and number of bacteria themselves. Whether or not such influences act by disturbing the bactericidal activity of the serum or by inhibiting the activity of the leucocytes is difficult to say. In the study just mentioned it would seem that the serum is the important factor. Other studies lead to a contrary conclusion. Thus a loop of colonic contents is more apt to produce a reaction than cultures of bacteria from the same material which contain many more bacteria. Fluid obtained from the peritoneum under these conditions shows a smaller number of leucocytes when the colonic contents are used. The explanation seems to be that the intestinal contents inhibit the activity of the leucocytes, possibly changing the reaction of the peritoneal fluid. Since information obtained from the laboratory is uncertain, obviously the details in concrete cases under clinical conditions are difficult to secure. The problem presents the phase of the subject about which information is most urgently desired.

The difficulties these problems present may well be understood by references to the literature. The great variation of infectivity became apparent early in the investigation of the etiology of peritonitis. Grawitz, who was the first to make a comprehensive study, found that suspensions of *staphylococcus pyogenes aureus* injected into the peritoneal cavity were quickly absorbed without harm unless there was a stagnation of peritoneal fluids or an abrasion of the surface. Burginsky, repeating Grawitz's experiments, discovered that the results varied much with the virulence of the organism. Cultures previously not virulent became so when repeatedly passed through animals and recultivated. These results seem to contradict my own studies already quoted. My cultures, it must be remembered, were obtained from the living animal or one just killed, while this author recovered his cultures from the animal dead of the infection.

Pawlowsky, evidently working with strains rivaling in virulence the reinoculated strains of Burginsky, found that very small amounts of bacteria were capable of producing a peritonitis. Wallgren likewise found that the degree of infectivity was due to the virulence of the organism and that the rapidity of the course of the disease was much influenced by the number of bacteria introduced. Finally Reichel's studies may be mentioned. He found

that while the injection of 2 to 7 c.c. of a gelatin culture of staphylococcus produced little or no disturbance, the injection of 100 c.c. of the same culture produced a hemorrhagic peritonitis in from 20 to 24 hours.

Without going further into detail it may be stated that sufficient evidence has been presented to emphasize the importance of virulence of a given strain of bacteria which gains entrance to the peritoneal cavity.

The evidence of the importance of the second factor in Grawitz's conclusions, namely, associated lesions, has been attested to by a large number of workers. Among these may be mentioned Orth and Waterhouse. These authors reporting on the same series of experiments found that rabbits, cats, guinea pigs, and rats could be injected without harm. The failure of deleterious consequences to arise was due to the fact that the bacteria were quickly absorbed from the peritoneal cavity. If foreign bodies such as agar or gelatin from the cultures were introduced, and particularly if blood was allowed to accumulate, much smaller numbers of bacteria sufficed to produce a peritonitis. Rinnie, Reichel, Walthard, Krafft, and Halsted showed that while a blood clot invites infection, in aseptic operations its presence may facilitate healing. Tavel and Lanz, Silberschmidt, and Wieland come to similar conclusions.

Noetzel, working with other bacteria, such as streptococci, *Proteus vulgaris*, *Bacillus coli*, and *Bacillus pyocyaneus* came to analogous conclusions. These, even more than when staphylococci were employed, were capable of producing peritonitis in the uninjured peritoneum. Because of the importance of the virulence of the strain this author concluded that the active resistance to bacteria was exerted *in loco*, according to him, due to the action of the peritoneal fluids and to a lesser degree to the action of cells. Wallgren ascribed an important role to the activity of the endothelial cells of the peritoneum and blood vessels.

**Varieties of Bacteria.**—Under clinical conditions the bacterial flora is nearly always complex. Nevertheless a recounting of the kinds encountered, together with a consideration of their characteristics when active in the peritoneum, so far as they have been determined, may not be without profit.

This is particularly true, for it may be stated as a general proposition, that those cases of peritonitis resulting from operation or from penetrating wounds of the abdominal wall are caused by the streptococcus or staphylococcus, except in operations involving the gut tract. Puerperal peritonitis is likewise usually caused by one of these organisms. On the other hand cases arising from spontaneous, operative or perforating wounds of the hollow organs are apt to present an astonishingly complex flora. I have isolated as many as a dozen different varieties from cases of diffuse peritonitis, the most of which, I may say, could not be identified.

**Streptococcus Pyogenes.**—This coccus, which is the most virulent if not the most frequent variety encountered in peritonitis, was first isolated by E. Fraenkel. He found this organism in ten of fifteen cases of peritonitis; in only two cases, however, was it the only organism present. Predöhl in the same year examined fourteen cases of perforative peritonitis in which four were due to pure cultures of this coccus and in four other cases this was the most prominent organism present. In four postoperative cases this was the only organism present, while in perforative cases a mixture of organisms was present. It is worthy of note that the observations of both authors just quoted were made on post-mortem material. A. Fraenkel likewise found the streptococcus prominent in 31 cases examined. In 5 cases of puerperal peritonitis it was the only organism present. Likewise Clivio and Monti found streptococci in pure culture in 5 cases of puerperal fever.

The characteristic biological features of this coccus make it possible that it is more often present than is recognized, because of its slow growth on artificial media and the likelihood, therefore, of its being overshadowed by more rapidly growing organisms. At any rate a small chain coccus is more often present in peritoneal infections when the exudate is examined on the slide than when plating is depended upon to demonstrate its presence. While this by no means identifies them, the fact that such cocci are most frequently found in those cases which run a course characteristic of the streptococcic infections lends probability to the suspicion as to the identity of organisms occurring in chains observed in smears.

The same holds true in the objective findings in the peritoneal cavity. Animals when inoculated with this organism respond by the production of a thin milky peritoneal exudate, not great in amount, associated with little or no cellular infiltration unless the organism is of attenuated virulence and the course of the disease prolonged. This organism is more apt to be found in the parenchymatous organs soon after inoculation than are other varieties. If associated with other organisms, as the staphylococcus or colon bacillus, the characteristics of the streptococcus will be masked and yet the deleterious effect of the streptococcus goes on unhindered. In the more localized infections the organism shows the result of its disposition to extend by way of the circulation. In the human subject, as will be noted in the section on "complications," abscesses in remote organs, notably in the liver, lungs, and joints, are of relatively frequent occurrence. Animals do not survive the disease long enough for these secondary foci to develop unless local areas of lessened resistance are produced by mechanical or chemical means.

**Bacillus Coli Communis.**—This is the common organism encountered in perforations of the terminal ileum and colon. As soon as the site of infection is reached the presence of this organism is announced by the characteristic odor. In cultures this organism quickly covers the media to the obliteration of other organisms. It is seldom the only organism present, however, though it is the most common organism encountered. Fraenkel noted the common association of this organism with the streptococcus. Malvoz was also one of the earliest writers to recognize the importance of this organism. Krogus found coli 35 times in 40 cases examined. Barbacci in 14 cases of perforative peritonitis found this organism 13 times. Tavel and Lanz call attention to the fact that because of the ease with which the colon bacillus grows on ordinary culture media it may cover up less readily growing varieties. They believe this because smears stained by Gram show many forms that do not appear in the culture plate. That the colon bacillus varies much in virulence is a matter of everyday observation. De Klecki, in experiments on dogs, believes he has demonstrated that in a loop of gut isolated by ligature a more virulent strain develops than is found in nonconstricted portions

of the gut. This may be conceded since any stasis promotes growth of bacteria. This same author believed that symbiosis with other organisms increased its virulence. Various American authors likewise emphasized the importance of the colon bacillus in the production of peritonitis. Among these may be mentioned Fowler, Hodenpyl, Park, and Richardson. In animals this organism is characterized by an abundant exudate, rich in cells and granular fibrin, leading to the agglutination of adjoining loops of gut. The exudate shows the cells which have escaped for the most part in a process of disintegration. My own studies are in accord with the conclusions of Tavel and Lanz. The colon bacillus alone is a relatively innoxious organism as compared to the streptococcus. In fact it seems to me that the presence of the colon bacillus limits the spread of a concomitant coccus infection because of the disposition of the colon bacillus to produce an abundant exudate which seems to counteract the negative chemotaxis (if one may still use this term) and lessens the absorption of the cocci. The earlier a diffuse peritonitis is analyzed the more apt is the streptococcus to be found, hence the surgeon is more apt to find it than the pathologist. The streptococcus likewise is more frequently found in the tissue than in the exudate, a fact which Rosenow has only recently emphasized.

Clinically the colon bacillus quickly produces a large amount of stinking milky pus intermingled with flocculi which when attached in large amount to adjoining loops of gut may produce an adhesion readily separated by the finger. The abundant cellular content usually shows varying degrees of degeneration.

**Staphylococcus.**—This organism is frequently found associated with other organisms, rarely alone unless the infection reaches the peritoneum from an adjoining region, as from a near-by abscess. It is more frequently associated with the colon bacillus than with the streptococcus. Dudgeon and Sargent ascribe to this organism a greater prominence than do most writers. According to them it is frequently present as a forerunner of other organisms. They even frequently find it in peritoneal cavities which at no time have been the site of active disease. Later in the disease when complete walling off has occurred and but a sinus remains, the staphylococcus is very apt to be found. In fact it may

be that the staphylococcus epidermidis albus may, like the bacteria of the soil, actually serve a useful purpose. A sluggish wound infected by this organism reacts, forming a fibrinous exudate which results in cicatrization.

**Bacillus Pyocyaneus.**—This organism not infrequently makes its appearance late in the course of an infection. It manifests its presence by the green-blue color of the pus. Its presence tends to produce a sinus which may discharge indefinitely.

**Mixed Infections.**—A pure culture is rarely found in peritonitis when it is the result of perforation of the gut tract. The above mentioned varieties in varying combinations furnished the most frequent organisms. Besides these may be mentioned the *Bacillus foetidus liquifaciens*, *Proteus vulgaris*, *Diplococcus intestinales*, and a host of organisms which even expert bacteriologists have failed to identify. Achard and Broca in 20 cases found mixed infections in 13, and v. Mayer found mixed cultures in all appendiceal peritonitides. Krogius in 40 cases found all but three to be mixed infections. This author found a large number of nameless forms. In 35 of his 40 cases he found bacilli with rounded ends with a vacuolated capsule which did not stain by Gram, which he could not identify.

**Anaerobic Bacteria.**—The presence of organisms on the smear which failed to develop in plate culture led numerous authors to suspect that anaerobic organisms might be present. Vellian and Zeber were the first to investigate this variety of organisms thoroughly. In 22 cases investigated they failed to demonstrate anaerobes in only one case. The species they isolated were as follows: *B. fragillus*, *B. ramosus*, *B. perfringens*, *B. fusiformis*, and *B. mucosus*. They blame the anaerobes particularly for those cases in which there is gangrenous perforation with the production of stinking pus. I do not believe this. Any organism that early involves the appendiceal artery may produce a gangrenous appendix.

**Specific Forms of Peritonitis.**—In certain forms of peritonitis a single bacterium is the active agent, resulting in a picture clinically distinct from those heretofore considered. Most prominent of these is tuberculous peritonitis. The gonococcus also gives rise to a clinically recognizable picture. As much may perhaps be said of the pneumococcus and influenza bacillus to which may possibly

be added the *Spirochete pallida*. The facts of interest relating to these organisms will be considered under the specific discussion of the disease they engender.

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## CHAPTER XII

### PATHOGENESIS OF PERITONITIS

Broadly speaking, peritonitis is the reaction of the peritoneum against any condition which destroys or threatens the integrity of any part of it. This effect, as we have seen, may be produced by the action of physical or chemical agents or by the products of bacteria.

Physical and chemical agents are of minor interest because reactions produced by them are limited by the duration and degree of their action and are incapable of producing a progressive disease, save as they may be associated with, or set up, bacterial processes. So infrequently is this type a matter of interest that it may be said that its importance is largely academic. However, the reaction to these agents has played an important role in experimental pathology and much of this has been repeated inadvertently by the surgeon in his therapeutic endeavors, mostly it may be added in the form of antiseptics, and by means that have been employed in the prevention of adhesions or for the control of hemorrhage.

The usual source of spontaneous chemical or physical irritation is the bursting of hollow organs normally free from bacteria into the peritoneal cavity. Among these may be mentioned the biliary and urinary bladder and the thoracic duct. The rupture of cysts permitting the escape of their contents may act in a similar manner. Among these may be mentioned cysts of the ovaries and less commonly parasitic cysts, notably echinococcus.

Peritonitis in its practical relations, however, we may say without equivocation, is due to the invasion of the peritoneal cavity by pathogenic organisms. This simple statement declares the fundamental factor, but the disease as manifest in the human subject presents the greatest variations due to the difference in the character of the organisms and the conditions under which they gain access. Because of the amazing range of these variations, so simply stated, this becomes the keynote to the proper understanding of the inflammations within the peritoneal cavity. Variations in species of bacteria and the vastly different degree of virulence they present is

complicated by the fact that various strains of the same order present no less a degree of variability. The complexity of the picture produced by this variability of the invading organisms is vastly complicated by the manner of their introduction into the peritoneal cavity. If the invasion is slow and the defensive forces have time to mobilize, the results are less disastrous than when the invasion is rapid and the infective agent enters the peritoneal cavity in the absence of an anticipatory reaction. In the latter instance the progress of the infection is unhindered. We have, therefore, two variables, both infinite in their range, and it does not require a mathematical mind to perceive that the product of these two variables is an infinitely inconstant picture.

Though this great variation in type of disease exists, it is possible to distinguish certain groups due to a similarity of causative organisms and the like circumstances in their introduction, as was noted in the chapter on etiology. It is the purpose of this section to define as closely as possible groups of like clinical course. It is only by possessing a clear notion of the fundamental factors that the surgeon is enabled to meet conditions in the most comprehensive way. While a sharper division is not possible, the desirability of the nearest possible approach is generally recognized by the disposition of the surgeon to designate the lesion according to the circumstances of its genesis and neglect the existence of the causative organisms, and even the presence of the overshadowing peritonitis, in his nomenclature. By this he bears testimony to the fact that a knowledge of the pathogenesis is necessary as a foundation for his therapeutic endeavors. As examples may be mentioned, appendicitis, perforated peptic ulcer, pericholecystitis, and pelvic peritonitis. All these threaten the patient with the same condition of spreading peritonitis, but he endeavors to separate them by designating the source of the infection rather than the nature of the disease itself. So different are these, grossly speaking, that the astute diagnostician is usually able to succeed in his endeavors at broad specific determination. While such endeavors are usually lacking in *finesse* it is by their consideration that we are guided with least error in our efforts to locate the source of the infection and so to divert from the patient the impending disaster.

While the examination of the fundamentals of the variables al-

ready noted can best be considered in the abstract, concrete discussions of diseases can not be avoided because only in this way can proper grouping of the otherwise too numerous factors be achieved.

The fundamental factors which govern the development of a peritonitis are simple. The well-being of the individual demands that the peritoneal cavity remain free from bacteria. Bacteria exist on all sides, being separated only by a wall of living tissue. The problem in pathogenesis is to study by what means bacteria gain access to the forbidden field of the peritoneum, and how they set up their nefarious business.

Any means that will penetrate the walls flanking the peritoneum may cause peritonitis. Bacteria are ubiquitous and those in the general environment of the body will gain access to the peritoneal cavity when there is a solution of continuity as by a wound, such as a gunshot, or a puncture or from the action of a disease. The solution of continuity need not be continuous. The bacteria may gain entrance at some distant point and be transmitted to the peritoneum by the blood or lymph stream. The first avenue is usually obvious, the latter in its very existence may be uncertain, even speculative. The superlative source of bacteria in peritonitis, however, are the hollow organs it covers and of these the gut tract is pre-eminent. Each of these methods of entrance may now be studied in turn.

**Penetrating Wounds of the Abdominal Wall.**—When there is a gross solution of continuity of the abdominal wall bacteria may be carried into the peritoneal cavity by the traumatizing agent, as by clothing carried by a bullet, or by some secondary agent, as the surgeon's efforts to determine the extent of the injury, or from some protuberant viscus accumulating infection from the environment and afterward retreating into the peritoneal cavity, either spontaneously because of changing intraabdominal pressure, or by manual replacement by the first aid attendant.

Penetrating wounds of the abdominal wall are astonishingly little likely to be attended by serious consequences. Every surgeon knows that injuries to the abdominal wall of even great extent are recovered from with surprisingly little disturbance, if a hollow viscus is not injured. This immunity from grave consequences is

due to the fact that bacteria, while existent in large numbers on every object, as compared to the intestinal content, are rare curiosities. Nor is this the chief factor. The bacteria of the external environment are in a large measure nonpathogenic, and even those of pathogenic heredity are so attenuated in their virulence by the unfavorable environment in which they have existed that they are but a minor menace to wound surfaces possessed of a normal capacity for combat.

Usually, too, large wounds are quickly sealed by natural protective factors. The abdominal contents, with the protecting omentum, quickly fall over the wound and by the time the blood flowing from the wound in the abdominal wall has had time to coagulate, adhesion of viscera to the abdominal injury has already taken place and further admission of infection is precluded. The relatively innocuous bacteria that have been carried in by the traumatizing force are quickly taken to account by the uninjured peritoneum of the remainder of the abdominal cavity.

It is only when bacteria of unusual virulence are introduced, or foreign bodies laden with bacteria remain in the peritoneal cavity, that an infective process gains foothold. The influence of the presence of a foreign body in aiding relatively innocuous bacteria in exciting inflammation has already been noted in the section on general pathology. In fact, generally speaking, all that has been learned in experimental peritonitis may be applied to the elucidation of the question under consideration.

**Hematogenous Infection.**—Under hematogenous infection we are wont to classify those cases of peritonitis the source of infection of which lies at a distance. Perhaps there is some basis for such a classification, for the peritoneum is sometimes involved in pneumonia, and it is but fair to assume that the blood or lymph stream has conveyed the organisms. These will be considered in a separate section. The same may be said of influenzal infections. There is much evidence that tonsillitis may produce an involvement of the lymphatic apparatus of the appendix with subsequent infection of the peritoneum. To what extent the blood stream may furnish direct transportation from some primary focus to the peritoneum is not known. In rare instances peritonitis follows infections by pus or other organisms in distant regions. I have seen diffuse

gangrene of the gut follow moist gangrene of an extremity. How the infection gains access to the peritoneal surface is not fully understood, most likely by producing thromboses in the vessels with subsequent rupture into the peritoneal cavity. The escape of bacteria by exudation through the unruptured peritoneum has also been assumed.

Cases are recorded in which a fatal peritonitis has followed erysipelas at a distance and peritonitis following scarlet fever has been recorded in a number of instances (e. g., Aubrée). Corollary to this are numerous cases of erysipelas of the cord in the newborn which are followed by peritonitis. Here the extension is no doubt direct through the walls of the hypogastric vessels. Moore made a collected report on this type. Additional cases are reported by Breton.

Even before the discovery of the streptococcus by Fehleisen, v. Leyden described a streptococcus in his case of peritonitis following erysipelas. Since the modern development of bacteriology, this organism alone or in association with others has been described, most frequently in alleged hematogenous infections.

That the bacteria reach the peritoneum by way of the bloodstream is likely. So far as the erysipelatos cases go it is interesting to note that Achalme got positive blood cultures in all of 13 fatal cases of that disease.

**Idiopathic Peritonitis.**—This caption is meant to include cases of peritonitis the origin of which is indeterminable. Despite every effort to discover the source of the infection, the best that can be done in certain rare instances is to secure ourselves behind such a classification. It is, of course, never a clinical classification because only the most painstaking autopsy can give the necessary exclusion of other foci to warrant its adoption. Though rare, occasionally throughout literature cases of peritonitis are recorded in which the source of the infection does not appear even after the most painstaking study. It is significant that these cases have become progressively less as knowledge and exactness of investigation have become greater. Nevertheless, cases occur in which there seems to be no detail lacking, yet the source of infection was not revealed. In such cases it is perhaps warranted to use the much maligned term "idiopathic."

The first case belonging to this group in which the data is fairly satisfying was recorded by Behier and Hardy. Bigelow recorded a case, the earliest satisfactory one in American literature. As an example of this type of disease the one recorded by Leyden may be cited. Following a diffuse diarrhea a generalized peritonitis developed which at autopsy showed no primary lesion. Meunier, and Milian and Harrenschmidt report two similar cases.

In all the cases recorded the disease was first manifested by a diffuse diarrhea followed by the signs of a generalized peritonitis. It would seem that the disease must therefore be a primary intestinal infection and that bacteria pass through the intestinal wall, a sort of discontinuous perforation as it were. In puerperal peritonitis such a condition commonly exists. I have had two cases belonging to this category. Both (females) began with chill followed by profuse choleric diarrhea, which was promptly followed by signs of diffuse peritonitis resulting fatally the fourth and sixth days, respectively. At autopsy there was a diffuse inflammation of the peritoneum, moderate exudate, some fibrinous deposits, and excessive intestinal distention. In one of these the distention was so intense as literally to fix the diaphragm. Each of these cases presented a streptococcus pyogenes and an unnamed diplococcus. Both of these were maiden ladies of middle life.

There is a suggestive sameness in the recorded cases corresponding in the main with these personal observations. Curiously enough, females alone seem to be affected. The significance of this, if it has any, does not appear.

It is possible that a general septicemia developed from some unknown source or that there was a primary bacterial dysentery. In this latter event, it is possible that there is a common source in such cases for both dysentery and peritonitis, though the autopsy does not reveal it. I have seen intense choleric diarrhea attend erysipelas of the face without peritonitis but with perisigmoiditis.

One needs to go back to the early literature on peritonitis in order to find any considerable number of case records. Surgeons now seem to question the propriety of recording cases of peritonitis the origin of which can not be demonstrated.

Guttmann in 82 cases of peritonitis described 8 cases as idiopathic. Litten believes that idiopathic cases occur, though they

are rare. Grawitz in 867 autopsies had 13 spontaneous cases. It may be possible that idiopathic peritonitis may occur from small local foci, the existence of which may be obscured by their location. It would differ from the hematogenous type then only by the fact that the focus is unknown.

**Perforative Peritonitis.**—Nearly all cases of peritonitis arise because of a perforation of some hollow viscus which constantly harbors bacteria. This perforation may result from violence as in gunshot injuries or other trauma or from surgical manipulations. In the vast majority of instances the infection escapes through a solution of continuity due to some disease process, which destroys the continuity of the wall of the gut. An ulcerous process may destroy the wall or an infection beginning in the wall may gradually destroy it, or the wall may become necrotic because of some sudden complete stoppage of nutrition to a certain segment, or such a degree of nutritional disturbance may exist that bacteria escape through a wall yet capable of regeneration and, finally, some form of local infection may rupture into the peritoneal cavity.

**Perforation of the Gut Wall by Mechanical Injury.**—A force acting from without may produce an opening into the gut permitting the escape of intestinal contents. When the opening is small, the elasticity of the submucosa may roll in the mucosa in such a manner as to actually close it to further passage of gut contents. Sometimes the great omentum becomes attached to the margin of the opening, effectually closing it, or several coils of gut may adhere about an opening and prevent the escape of the contents.

The usual fate of a perforation is to permit the escape of gut contents consisting of fecal masses and bacteria. As noted in the section on general pathology it is the mechanical irritation of the gut contents that plays an important part in producing the most favorable conditions for the development of the bacteria that escape from the gut. A spreading peritonitis, therefore, is the rule when a gut is perforated permitting the escape of the contents.

**Perforation by Ulceration.**—In this condition there is a solution of continuity of the walls of the hollow viscera as a result of disease or foreign bodies. The virulence of the infection resulting is dependent on the rate of development of the ulcerative process and the degree of reaction which takes place in the peritoneum in this



region as well as in the surrounding tissue. Because of these differences, infections resulting from perforations may be divided into those which occur without reaction, those with reaction in which inflammation occurs in the gut wall in the region of the perforation, and those in which preliminary adhesive inflammation takes place about the site of impending perforation which produces protective adhesions.

**Ulceration without Reaction.**—In some instances the process is so rapid that the continuity of the wall is so quickly lost that there



Fig. 155.—Perforating ulcer of the duodenum. There are no adhesions about the opening.

is no reaction. Rapidity of the process must be due at least in part to the environment, for this condition is noted chiefly where the ulcer is bathed in digestive juices, notably in duodenal, typhoid and similar ulcerations. The gall bladder and cecum sometimes perforate in a like manner. The occlusion of vessels in a limited area seems to be the factor at fault in some instances. These are thrombotic perhaps rather than ulcerous perforations.

A sharply defined ulcer results, the edge of which presents without reactive infiltration of any kind. These are well deserving the

clinical designation of "punched out" ulcers (Figs. 155 and 156). They are really perforations, lacking the attributes of ulcerous processes. When perforations of this character exist there is an unobstructed pouring out of the gut contents preventing any effective attempt at walling off. However, the absence of adhesions is due not alone to the intensity of the bacterial invasion, but also to the pouring out of the digestive ferments which prevent the

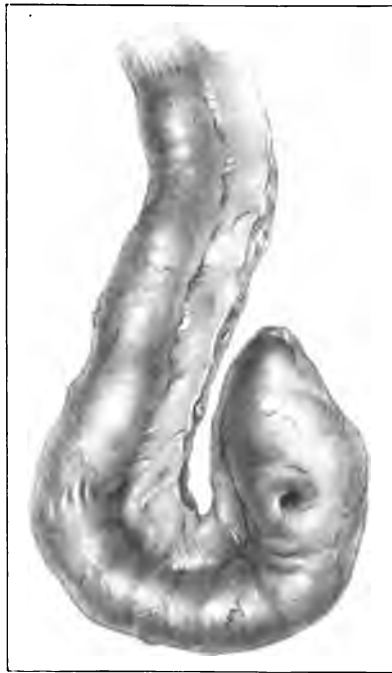


Fig. 156.—Acute perforation of the appendix. The whole organ was black and the site of perforation represented an area which first became liquefied.

fibrin formation so that any attempt at the development of adhesions is prevented. This is seen in a pronounced degree in perforations of the duodenum in which the gut contents pour out unhindered.

**Ulceration with Reaction.**—This condition is typified by the "indurated" ulcers of the stomach and in some types of appendicitis (Figs. 157 and 158). Here the solution of continuity is attended with marked, often enormous thickening of the wall of the viscus.



Fig. 157.—Inflammatory thickening of the appendix about an enterolith with a perforation proximal to the foreign body.



Fig. 158.—Slight inflammatory thickening of the appendix with perforation at the tip.

This thickening is an expression of the attempt at healing. It is in a large part made up of round-celled infiltration and fibrinoid exudations, and to a lesser extent to the new formation of fibrous tissue.

When perforation occurs under these circumstances the attempts at repair are exceeded by processes of dissolution until the entire thickness of the wall is destroyed. While there has been no formation of adhesions, the reactive process on the part of the peritoneum may be pronounced and in consequence walling off processes are instituted more readily when therapeutic endeavors are instituted. Since the visceral contents escape unhindered, the clinical results are the same as the previous type of perforation unless aid is offered by operative means. In this type the individual may have been disturbed by preliminary manifestations absent in the other type.

The majority of perforations belongs to this type. Aside from the ulcers of the stomach already mentioned, it is noted in varying degrees in typhoid ulcerations, in tuberculous and malignant processes as well as in appendiceal and gall-bladder infections, diverticulitis and the like. In this type as in the "punched out" variety, there are no adhesions present, but the surrounding tissues have already reacted and in response to additional irritation may quickly do so.

**Ulceration with Adhesion Formation.**—In this variety as the ulcerous process approaches the surface the peritoneal tissues are set into a state of reaction. By virtue of this reaction an exudate is thrown out which attaches the threatened surface to its environment. In this way an artificial reinforcement is produced so that when the peritoneum is finally destroyed some neighboring part is attached firmly to the gut which prevents escape of the contents (Fig. 159). This state is noted most frequently in appendiceal perforations and may exist about stomach ulcers, less often about duodenal ulcers, very occasionally about typhoid ulcerations. Not infrequently perforation of the gall bladder is prevented by the attachment of the ubiquitous omentum.

Often the protective adhesions are but partial. In that event a local peritonitis or abscess may form, or the barrier may be passed because the adhesion does not form a perfect wall, and the infection

may spread. When the adhesion formation becomes complete, the infection forms a localized abscess and its contents may be conducted to the parietes and the infection may ultimately involve this structure and a fistula result. This is sometimes noted in gall-bladder infections, less often in perforations of the gut tract. More often the localized abscess breaks into the lumen of the gut from

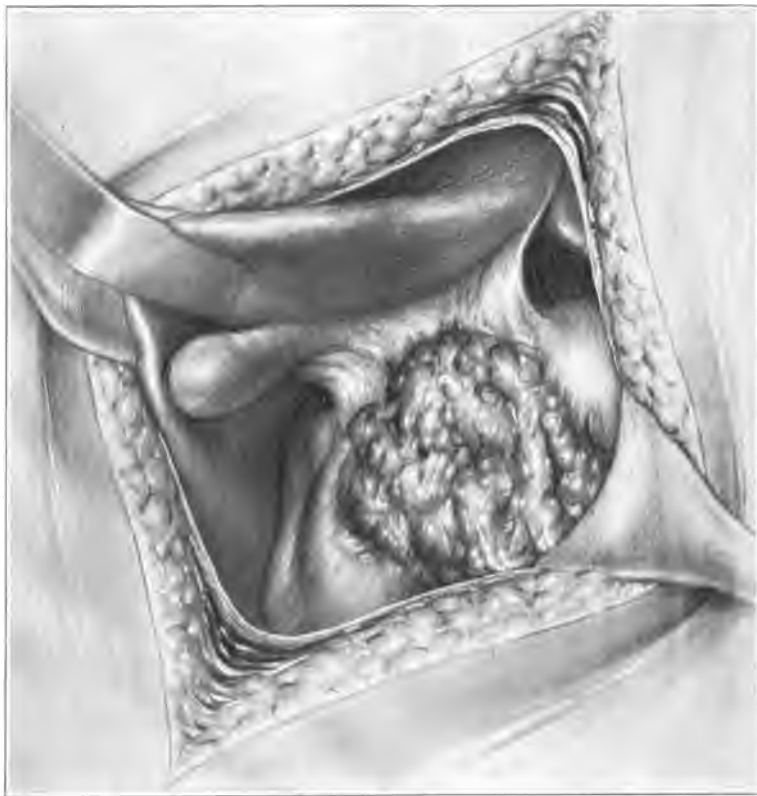


Fig. 159.—Ulcerating duodenum in which perforation was prevented by the formation of omental adhesions.

which the infection originated or into some other hollow viscus.

**Infection within the Wall of the Gut.**—How often this may take place in ulcerations can not be determined. Here the infection gains access to the wall of a viscus and by extension involves both the mucous and serous surfaces. In this way the entire thickness

of the gut wall is destroyed and an unhindered passage for the escape of contents is provided.

The consequences of this type of lesion are very varied. In many the reaction produced in the surrounding peritoneum is so intense that protective adhesions result (Fig. 160). The abscess may then break within the lumen of the gut and the area is drained and in due time only the scar in the gut wall and perchance an adhesion is all that marks the previous existence of a menacing lesion.

The virulence of the infection may be such that the formation of adhesions is repelled and the wall is perforated permitting the unhindered escape of the gut contents.

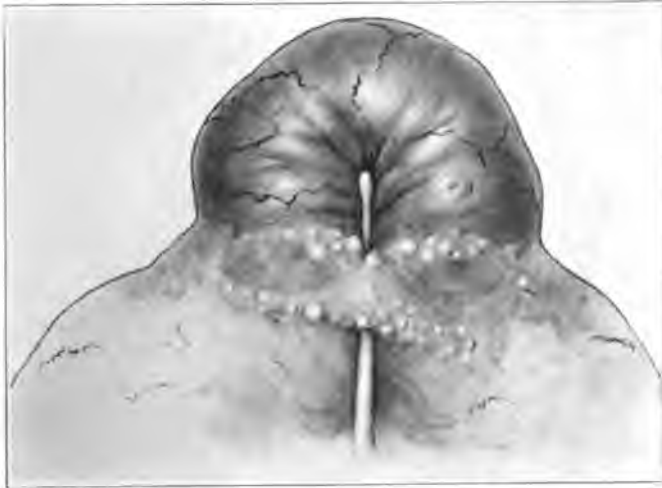


Fig. 160.—Small abscess within the gut wall covered with plastic exudate. A probe has partly separated this exudate. From a case of irreducible inguinal hernia.

There may be a midstage between these two conditions and a localized abscess is formed, or the partial adhesions may serve as a gubernaculum for the infection and an abscess may finally result.

This variety of affection is most often demonstrated in the appendix, but may occur anywhere in the gut tract. The infection takes place in the lymph follicles of these organs and by extension affects the mucosa as well as the muscularis and serosa. Sometimes several lesions may be found in the same gut, representing varying degrees of development, permitting the formation of some

opinion as to how the process advances. It may also be found in the bladder wall, within the tube or uterus, rarely in the wall of the stomach. Abscesses in the solid viscus may reach the peritoneum in a like manner, as abscesses of the liver.

**Infection by Stasis.**—When the circulation of the gut wall reaches a certain degree of embarrassment it becomes pervious to bacteria. This may take place before the wall is injured beyond restitution if the disturbing process is removed, as is sometimes observed in strangulated hernia (Fig. 161).



Fig. 161.—Necrosis of a loop of ileum in a case of strangulated femoral hernia.

To determine what character and extent of lesion must exist before it is possible for bacteria to escape many studies have been instituted. When there is an obvious solution of continuity or a state of degeneration of the gut wall which precludes a return to the normal, the problem seems simple enough, for an actual perforation results. In some instances, however, there is no solution of continuity and the gut wall is not so far diseased that it can not become restored to the normal, yet bacteria pass. This factor is most clearly observed in strangulated hernias in which bacteria

may be demonstrated in the exudate, but in which there is a complete recovery after the release of the strangulation.

That a primary infection of the gut tract is not necessary to produce these changes which will permit the escape of bacteria is probable from the fact that in degenerative ovarian cysts, which come in contact with the gut wall, so irritate it that an infection of the cyst takes place, due presumably to bacteria that have escaped from the gut canal. Even the blood clot from an extra-uterine pregnancy that has ruptured may so act. Olshausen ascribed the infection of ovarian cysts to direct extension from the gut tract. Walthard attempted to prove that more than adhesion of serosa to serosa was necessary before bacteria would traverse the gut wall, a fact abundantly proved by clinical experience.

The means by which bacteria travel in such instances evidently is that a fibrinous exudate forms within the gut wall as well as upon its surface. A like condition exists in the cyst wall or blood clot. These two are connected by fibrin masses. In this way a homogeneous structure results which permits bacteria to pass.

That bacteria may escape with the exudate into the peritoneal cavity in intestinal obstruction has long been known. Nepveu was the first to make this observation. Garré found a coccus non-pathogenic to animals in only one of eight cases of intestinal obstruction examined. Naturally the likelihood of bacteria being present is dependent upon the degree of injury to the gut wall. Clado found the colon bacillus in three out of five cases examined. All three of these in which the bacilli were found proved fatal. That bacteria may escape early was proved by Bönnecken's case in which he found bacteria after strangulation was present only 4 hours. Lanz and Tavel in 21 cases of strangulated hernia found bacilli in five. Rodella secured negative results by both aerobic and anaerobic cultures in all of 5 cases. Dudgeon and Sargent examined 47 cases. In forty-two of these the fluid obtained from the sac was sterile, in 5 bacteria were present. The colon bacillus was found but once in pure culture, once with the staphylococcus albus, and once with a Gram-positive staphylococcus. Anaerobic cultures were made in eight without demonstrating any anaerobes.

Numerous observers have attempted to solve the problem of how



bacteria escape by artificially induced intestinal obstruction. Reichel found that even after an ileus of several days' duration the gut was not pervious to bacteria. Attempts at imitating the condition in strangulated hernia were made by Zeigler and Tietze. These experimenters placed a loop of gut in a condom and constricted the base by a ligature. The former in 29 experiments found bacteria 9 times in 17 cases. Wurtz and Hudelo found bacteria in the peritoneal exudate in one-half the cases in animals killed during acute alcoholic coma. The presence of an extensive peritonitis chemically induced is insufficient to insure the transudation of bacteria. E. Fraenkel induced intense reactions with iodine and chloride of iron and found that bacteria escaped from the lumen of the gut only after destructive changes in the gut wall had taken place. Tuffier allowed urine to escape into the peritoneal cavity and found that bacteria did not escape from the gut until profound changes had taken place.

The escape of bacteria into the general peritoneal cavity may occur either from the vessels or from the gut tract. The protecting barrier is the Kittsubstanz between the cells. At least I so conclude because a precipitation of that substance with silver nitrate increases the permeability of the tissue. For instance if a dilute solution of silver nitrate is injected into the mesenteric vessels, bacteria-containing fluids will escape under relatively low pressure into the peritoneal cavity, while if not preceded by the silver injection much higher pressure is required. Binaghi concluded that it was the peritoneum that protected the peritoneal cavity from the infection from the intestinal cavity. From my own studies just noted it would seem that these conclusions are sound but it does not furnish the sole barrier, for intestinal walls devoid of peritoneum do not become permeable without some change in the remaining layers.

When a gut wall becomes so changed that an actual solution of continuity takes place it goes without saying that an escape of contents will take place. If an area of gut wall loses its vitality through interference with its circulation or by cauterization with a strong chemical sufficiently great to destroy the vitality of the tissue, escape of bacteria will take place before the diseased portion of the gut is cast off. The protection the destroyed gut offers seems

to be purely a mechanical one. If a hypertonic solution is placed external to such destroyed gut, bacteria will escape sooner than if the diseased gut is surrounded by an isotonic solution. In an injured gut the escape of bacteria takes place in the zone of viability first, that is, in that area where the reaction of living tissue is taking place against the dead. In a constricted gut the greatest number of bacteria are found near the line of constriction and in a stained section the greatest number of bacteria will be found in the tissues of that region. Tissues which have undergone changes to the degree that they are no longer acidophilic are most likely to harbor bacteria, in fact it is the only living tissue in which bacteria are found. In such tissue if the current is reversed by any factor, bacteria escape. If a gauze pack is laid against the inflamed gut the irritation from the foreign body will cause a current of fluid to set in. This fluid will likely contain bacteria while the fluid found on the surface of the gut at some distance from the pack will contain none. Every surgeon knows how prone a drain placed against an inflamed organ is to produce a fistula from that organ. The production of a fistula is but one step removed from the reversed current which carries bacteria to the drain. Non-irritative substances such as glass and rubber are less prone to produce fistulæ than is gauze or other irritating substances.

From these studies it seems a safe hypothesis to assume that for bacteria to escape from the gut lumen into the peritoneal cavity two factors must be present: disturbance in the intercellular substance, the result of some reactive or chemical process, and the action of some factor to reverse the current of absorption.

Reaction from other causes than interference with the circulation may make the peritoneum permeable to microorganisms. Gonococcic tubes permit the passage of infection after the fimbriated end has been occluded. The puerperal uterus presenting septic thrombi within its walls likely does the same thing. In fact any infection lying near the peritoneum may give off its infecting material under certain conditions. It is, therefore, not entirely correct to class all these conditions under stasis since disturbance of circulation is but one of the factors active. There seems to be no more accurate term, however.

**Infection by Necrosis.**—As a corollary, sometimes as a sequence,

to the preceding there is a total solution of continuity from degeneration of the gut wall. This form is closely allied to perforation by ulceration without reaction but differs in involving a segment. Gangrenous appendicitis and gall bladders properly belong here.

This variety of affection may show anywhere that a vessel may be closed. Occlusion of the appendicular artery with total necrosis of that organ is the most common example while perhaps the most striking picture is seen in mesenteric thrombosis (Fig. 162). In the former usually there is a primary infection of the wall of the appendix which extends to the artery, producing within it a septic thrombosis. In the case of the mesenteric thrombosis the occluding agent travels from afar and occludes the vessel but some more local process may produce a septic thrombosis.



Fig. 162.—Necrosis of the appendix from thrombosis of the appendicular artery.

Anywhere that the circulation may be destroyed this process may take place. I have seen a segment of the transverse colon fall out from necrosis produced by extension of infection from a gall bladder. The small gut may suffer a similar fate when it lies near a tumor undergoing necrosis.

**Perforation of Paraperitoneal Abscesses.**—Any abscess arising external to the peritoneum may escape into the abdominal cavity. Abscesses of the abdominal wall may perforate the peritoneum. I once saw this accident occur in a child of four years.

These are among the rarer accidents in abdominal diseases. They must be differentiated from walled-off abscesses within the peritoneal cavity. Abscesses occurring within the parenchymatous organs, liver, uterus, spleen, etc., are sometimes included under this head.

**Location of the Infection at a Distance from Its Source.**—In some instances the infection may escape at one point (Fig. 163) and be conveyed to some distant point and there set up the chief reaction. It is necessary in such instances, when there is not a suffi-

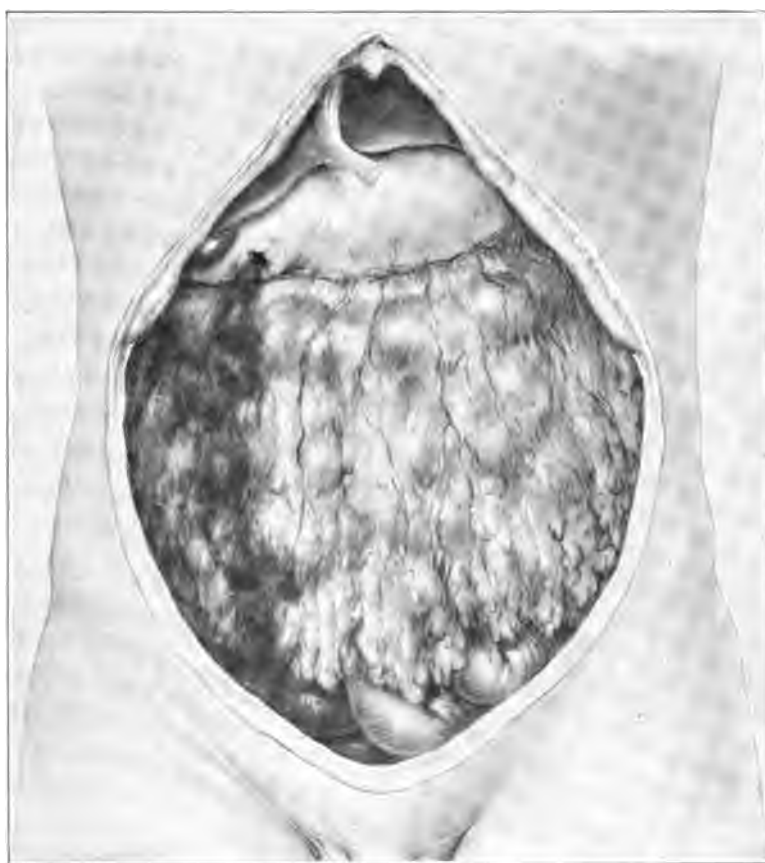


Fig. 163.—Perforation of the duodenum showing how the contents of the gut are conveyed into the pelvis laterally to the colon.

cient pathologic change in the region to account for the degree of infection, to seek further for the source of the trouble. This condition is encountered particularly in perforations above the attachments of the great omentum.

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## CHAPTER XIII

### GENERAL SYMPTOMATOLOGY OF PERITONITIS

Notwithstanding the great variety of manifestations in the various cases of peritonitis the fundamental phenomena have much in common. It seems best, therefore, to give a somewhat detailed account of the fundamental symptoms as observed in an average case of acute diffuse peritonitis. It will be easier then to consider the special forms which present peculiarities in one way or another because of the site of their origin.

Though the symptoms about to be detailed may occur in varying orders or more or less simultaneously, the presentation of each group of symptoms as entities much facilitates the presentation of a very complicated disease. The most common order of the occurrence of the symptoms may be followed.

**Pain.**—The perception of unpleasant or distressing sensations by the patient is the cardinal symptom in peritonitis from whatever cause. In the chapter on physiology the question of sensibility of the peritoneum was considered. That the parietal peritoneum may transmit painful impulses goes without saying. That the visceral peritoneum also may do so can hardly be denied.

Because of the two types of nerves which supply these tissues two types of phenomena must be recognized. The initial pain in peritonitis is usually that transmitted from the diseased organ to the region of the semilunar ganglia. Distinct from this pain is that produced at the site of the inflammation due to the irritation of the sensory nerves by the reactive process. We may speak of these as the reflex and reactive respectively.

**The Reflex Pain.**—When the causative factor is within the wall of the gut tract the pain is apt to be felt in the region of the epigastrium. This is true only so long as the disease process is confined to the wall of the viscus and does not reach the peritoneum. The reason for the reflection toward the epigastric region is that this is the region of splanchnic nerve convergence. This is really

understood when the embryonal migration of the organ is remembered. The character of this pain is similar to that elicited by traction on a loop of gut or by distending a gut with fluid or stretching its walls with forceps. In disease it is probably due to the stretching of the Meissner and Auerbach plexuses by the inflammatory edema.

**The Reactive Pain.**—Why the pain later becomes localized in the region of the lesion is a matter of controversy. At the suggestion of my teacher, Professor H. Virchow, I made a series of dissections seeking to locate the central termination of the nerve supply of the gut tract, and the relation of the sympathetic ganglia to the root ganglia. About all that can be said is that the nerves supplying the gut wall terminate in the semilunar ganglia and those spinal nerves supplying the abdominal wall are connected with these ganglia through the rami communicantes. There is no warranty for supposing that any spinal root ganglion is continuous with any particular division of the sympathetic. The fibers simply can not be traced with anything like such accuracy.

The only reason we have for assuming that the primary pain is recorded in these ganglia is that the pain is referred to their general location, and that severe primary prostration may occur in severe visceral disease which resembles closely primary injury to these ganglia.

The popular explanation therefore which assumes that the parietal pain is located where the cerebrospinal nerve is distributed which is connected with the ramus communicans of the sympathetic coming from the site of the lesion is without scientific basis. The stock argument in favor of this view is that no matter where the appendix is located the pain and tenderness in the abdomen is felt at the same region corresponding to the termination of the parietal nerve. This is a very pretty "library" theory but there is little to substantiate it clinically. Splanchnic nerves go from the region of the appendix to the semilunar ganglia. Fibers go from these ganglia to the root of the twelfth intercostal ganglion or thereabouts. The theory is weak in at least two particulars. It is impossible to trace fibers from the region of the appendix to the twelfth spinal ganglion. That they go there is pure hypothe-  
cation. The other error is that if pain were due to transmission



of impulses from the ganglion to the twelfth nerve the pain should be felt in the nerve terminals instead of somewhere midway. Why then is the pain felt at the same point irrespective of the location of the appendix? The answer is simple! It is not. Pain derived from irritation of the parietal peritoneum, spontaneous or from pressure, is felt at the point of irritation. The pain in appendicitis for instance, is usually at or near McBurney's point because that is where the appendix *usually* is. If the appendix is not here the pain is where the lesion is. The same is true of other affections, as of the gall bladder. While the base of the appendix, the usual site of the inflammation, usually varies but little from a fixed point, it does vary and sometimes greatly so, and when there is a deviation from the fixed point the site of pain varies also. I have seen an appendicitis in the left inguinal canal in a case of cecum mobile, the pain was all in the canal of the left side and the right side was free from pain. The diagnosis of strangulated omental hernia was wrong, but justified; in another the appendix formed an abscess between the sigmoid and uterus, simulating salpingitis, but the tubes were free. There was no pain in the normal site of the appendix. I have several times mistaken an appendicitis lateral to the ascending colon for a cholecystitis. In all of these cases the pain and rigidity was where the lesion was. The same rule applies to peritonitis from any other cause, as in impending perforation. A very long inflamed gall bladder may cause pain at some distant point from that usual in inflammations of this organ. I once saw the late Professor Koenig operate for a supposed appendicitis and find the fundus of the gall bladder attached to the abdominal wall in the ileocecal region.

Strangulated tumors cause pain where they exert their irritating effect upon the parietal peritoneum irrespective of the relation of their nerves on the organ from which they are derived, and irrespective of where they obtain their pedicles.

Pain in its strict clinical considerations may be divided into two categories, that produced spontaneously by the action of the disease itself and that elicited by the pressure of the examining finger or by the movements of the patient, as of the psoas in lifting the thigh in appendicitis or the movement of the diaphragm in cholecystitis.

**Spontaneous Pain.**—If a deeply lying process gradually approaches the surface pain may begin gradually and grow in intensity as the area involved increases. If, on the other hand, there is a sudden escape of irritating substances, as in the perforation of an organ, pain is sudden and intense and the most emphatic adjectives in the language are employed by all nations in describing it. Dreadful, awful, indescribable, are the common terms applied by those who have experienced its pangs and have lived to recall the experience in the calmer moments of restored health.

The pain does not remain in its fullest intensity but remissions and exacerbations occur until the phenomena of inflammation become established, then it tends to become more constant. The reason for this variation in intensity is the succeeding contraction and relaxation of an inflamed organ and the intermittent outpouring of the irritating substance.

The intensity and character of the initial pain varies somewhat according to the organ and pathologic conditions. Sudden occlusion of the appendicular artery with subsequent complete necrosis of the appendix is attended by sudden severe pain. The reason for this is not clear. In the case of the appendix the early chemical changes of impending grave nutritional changes probably exert an irritating effect upon the nerve terminals and it ceases when the organ has undergone a degree of degeneration destructive of all nerve conductivity. After this has occurred the appendix in this state acts as a foreign body and may irritate the parietal peritoneum and a certain degree of pain remains, but it may be absent in the presence of complete necrosis. I have more than once hauled a long black appendix from the depth of the pelvis from patients who had been quite free from spontaneous pain for some days.

In duodenal or gastric perforation the cause of the intense pain is yet more difficult to explain. In the very early cases, at the very beginning of the intense pain there is as yet no evidence of reaction on the part of the peritoneum and we must assume that the pain is due to the direct irritation of the nerve endings in the parietal nerves by the escaping fluid. I once placed a small drop of this duodenal fluid on my conjunctiva and severe pain was produced instantly. I once saw a perforated duodenal ulcer less than an hour after the initial pain. There was an escape of a few clots

of milk and a considerable amount of gastric contents. There were no visible inflammatory changes on the surface of the peritoneum. The pain ceased largely after the hole was closed. I have been able to determine, from the study of patients under local anesthesia, that pain is produced by irritation of the parietal peritoneum by dilute hydrochloric acid. In perforations which have existed some hours the pain is often over and lateral to the ascending colon. This distribution could be the result only of parietal nerve irritation.

On the other hand, large exudates sometimes are attended by little or no pain though the abdomen is filled with pus. These patients sometimes consult the surgeon in his office presenting other phenomena of severe generalized peritonitis. I once had a young man come to my hospital in a buggy. He had had soreness in his side for a week but for two days had felt more comfortable and save for weakness and an increasing dyspnea he believed himself better. He presented extreme meteorism and as I reached to count his pulse I was shocked at the clammy feel of death. The autopsy done a few hours later showed a large amount of free purulent fluid from a partly walled off appendiceal abscess which had ruptured.

These exudates exert little irritation and there is little reaction on the part of the peritoneum. They are analagous to large bouillon cultures as it were of virulent bacteria. Toxicity evidently has nothing to do with pain. Abscesses rupturing secondarily into the peritoneal cavity are often attended by a distinct sense of well being, until distention and creeping pulse awaken one to the disillusionment.

When pain is due to inflammatory reaction of the peritoneum the onset is more gradual and constant and the pain increases in intensity until the highest point is reached and then gradually lessens. The sources of pain in this common infiltrative variety of peritoneal reaction most likely are several. The primary pain is most certainly due to nerve irritation from the chemical exudate into the tissues. It increases as the exudate increases. Contraction of hollow organs may add to this early in the disease. Very soon, however, the gut wall becomes paralytic, probably reflexly, and this type of pain ceases. These cramp-like pains occur before the ordinary reactive phenomena of inflammation have had

time to establish themselves. After inflammation has become established the cellular exudation about the nerves no doubt plays the same role as inflammation in other tissues, namely, irritation of, or pressure on, the terminal end-organs.

Localized or localizing abscesses may be the site of great pain due to pressure upon the tissues. At least this seems a fair inference since pain ceases when such an abscess is relieved of its tension either by incision or spontaneous rupture. Sudden return of pain spells perforation into the free peritoneal cavity in many instances where a walled-off abscess had previously been established.

**Pressure Pain.**—Under this head may be included all agencies which change the mechanical relations of the different parts involved in the inflammatory reaction. Compression from without either accidental, as contact of bed clothes, or designed, as the manipulations of the surgeon, may be contrasted with the voluntary or involuntary movements of diseased parts against each other as in breathing or coughing.

It is not clear in just what way pain is increased by these acts. Mechanical pressure of the exudate is believed to be one of the causes of the increased pain. The rubbing together of inflamed surfaces as in pleurisy may be another source of pain. In assuming that increased pressure may augment the pain, we must meet the paradox that in local anesthesia pressure is supposed to be one of the factors which produces anesthesia. The differences in the character of the fluid and the state of the tissue doubtless are the determining factors. There are so many factors present in inflamed tissue which so change the argument that it is not necessary to impeach the arguments of local anesthetists.

In the presence of inflammation of the peritoneum there sometimes is exquisite hypersensibility of the skin. This is due to irritation of the nerve trunks in continuity and consequent hyperirritability of the end-organs in the skin. That the nerve trunks are affected as they course along the internal rectus and transversalis may often be observed in operations when an extensive edema affects all these layers. With the nerve shafts so buried in exudate, hypersensibility is easily explained. Deeper pressure may affect the hydrostatics of the deeper tissues and thus increase the

pressure on the nerve shafts. Pressure which depresses the entire thickness of the belly wall irritates the end-organs of the parietal peritoneum.

That friction is an important factor is well illustrated by the movements of the patient and by the movements of respiration and even more violent movements of coughing and vomiting. The surgeon is able to use the spontaneous attempts at fixation in clinical diagnosis. He groups these phenomena under the general head of muscular rigidity.

That pain is produced by the mechanical contact of one diseased surface against another is further attested to by the attitude of the patient. He tends to limit its production by assuming a position that will reduce the movements of the affected parts on one another, and any pressure from without, to a minimum. Flexion of the thigh in appendicitis or costal respiration in peritonitis having its seat in the upper abdomen bears such mute witness. In this category belongs the protective fixation of the abdominal muscles. Though the patient is not aware of it he makes use of his voluntary abdominal muscles to protect the affected parts beneath. These are quite as purposeful, if less voluntary, than is flexion of the thigh. The extent of muscular rigidity is dependent on the extent of the disease. Usually that segment of the abdominal wall covering the lesion alone is set in spasmodic rigidity. This regional fixation is possible by virtue of the inscriptions tendential. These scar-like bands dividing the rectus muscles make it possible for the abdominal wall to act in as many segments as there are segments produced by these inscriptions.

The patient may complain of equal pain in all regions of the abdomen. By producing pressure alternately in various regions the surgeon is often able to determine the seat of trouble by the facility with which he can increase the pain in some regions as compared with others. The cause of this heightened pain by pressure has already been explained as due to the pressure of one inflamed surface against another. After the disease has existed some days fallacies may creep in in certain cases. With increasing exudate the pain at the site of greatest inflammation may lessen because the fluid exuded keeps the affected surfaces apart. Analogous conditions are produced when an exudate forms in pleurisy. Sometimes

when there is abundant exudate there may be but little pain and consequently little rigidity. It is not uncommon to find extensive acute inflammation at operation when there has been but little pain and uncertain rigidity for some days before operation. In these cases there is usually considerable exudate. The inflamed parts float in an isotonic fluid, toxic though it may be.

The character of the inflammatory processes has much to do with the signs of rigidity that may be elicited. In the beginning as noted, there may be superficial hypersensibility with generalized equal muscle rigidity. As the disease progresses this phase is more marked and gradually becomes limited to the area involved. Because of this early in the disease it may not be possible to detect the site of the lesion by physical examination but one must await the relaxation of the uninvolved areas before the offending area can be detected by the persisting rigidity. When the limitation of rigidity occurs the inflammation in the surrounding parts has regressed as the tissues immediately around the infective focus show their ability to cope with the disease.

Unfortunately muscular rigidity spells extent of reaction, not gravity of disease. When, for instance, complete necrosis exists nerves may be lamed and physical examination may fail to elicit premonitory evidence of impending separation of the devitalized parts which presage the opening of the lumen of the gut.

Very violent infections which repel reactive factors in equal proportion fail to produce pain. In these conditions the patient may die of toxic absorption without there being either spontaneous or elicitable pain. In these cases, however, the pinched look and the glassy eye cause spontaneous contracture of the surgeon's own muscles as he involuntarily shrinks from the scene of impending disaster.

Taken all together, this element of pain is the great signpost of peritoneal inflammations. Spontaneous pain, its location, its manner of onset indicates very closely the organ involved, to him who understands how to secure a sequential story. Coupled with it the trained finger by detecting the niceties of variation in muscle tension is able to outline the battle field no matter what the source or character of the offending agent.

The young surgeon must learn fully these two elements of spon-

taneous and elicitable pain. By reading case reports of the masters he may learn much of the manner of onset of these inflammations, but it is only by careful palpation of inflamed abdomens that he can educate his fingers to recognize the story the muscles tell. The latter is the more important, for their veracity is unimpeachable. If these lessons are not learned, all the laboratory tests known to science will not guide him past the pitfalls of error.

**The Gastrointestinal Tract.**—That the digestive tract should share a heavy part of the affections of the peritoneum is easily understood when the topographic relations are remembered. To this must be added the intimate relation of the nervous apparatus of all parts of the gastrointestinal tract.

Involvement of one region of the peritoneum may reflexly set in motion reflex contractions of remote parts at the onset of the disease. This gives rise to one of the cardinal symptoms, vomiting. Later reactive processes may limit the movements of the entire gut tract, also reflexly, and we recognize paralytic distention. When there is extensive involvement of the gut wall the nerve plexuses become involved and the muscle coats are rendered incapable of contraction. Finally when inflamed areas come in contact they become agglutinated and movements may be mechanically hindered.

**Vomiting.**—Early emesis gives no evidence whatever of the location of the disease. This most likely occurs reflexly through the sympathetic system. The stomach nerves become sensitive, as is evidenced by increased vomiting when fluid is taken into the stomach. A direct irritation of the musculature may take place in some instances. Vomiting is one of the early symptoms, following immediately the advent of pain. Nothnagel says that vomiting may precede pain. So it may in exceptional cases, though it may be emphasized that this is not the rule. After the disease becomes localized, vomiting usually ceases. Vomiting at this stage is of no great prognostic significance. Recurring later it is of the gravest moment for it frequently indicates spreading inflammation or dynamic ileus.

Belonging to this phenomena is hiccough which appears most often late in the disease when the diaphragm becomes involved. This reflex irritation is sufficient to overcome the fixation of the

voluntary muscle for the sudden contraction of the diaphragm is often very painful to the patient.

Early vomiting is expulsive forcing from the stomach of whatever may have been taken into it. Later mucous or bile and in rare instances blood is expelled. Later, particularly in the moribund state, mouthfuls of fluid are expelled at frequent intervals apparently without much effort. The mechanism of this late vomiting is difficult to explain. Irritation of the phrenic terminals probably is responsible.

Vomiting of blood is noted in rare instances. Gerassimowitsch records eleven cases. In seven microscopic examination of the stomach wall was made. There was round-celled infiltration about the tips of the glands with enlargement and abscess of the lymph follicles. The nuclei of the gland cells did not stain.

**Meteorism.**—Distention of the gut follows the lesion of the gut wall. Disfunction of the muscle or nerves may be at fault. The distention is sometimes reflex for not infrequently tympany is observed in regions not the site of inflammation at all. Similar phenomena are noted in injuries of the back in which great distention of the gut may occur when there is no thought of a peritonitis. No lesion of the nerves can be demonstrated in such cases.

The changes that take place in the gut wall have been detailed in the section on general pathology. It may be repeated here that usually more or less of the entire gut wall is edematous and infiltrated. In the lesser degree the muscle fibers may show no change, or at most, fine granular degeneration. In cases where there is a more extensive degree of involvement the muscle cells may show a distinct paleness in structure and in that type in which the gut wall is much thickened and covered with a greyish exudate the muscle fibers may refuse all dyes. In this type of degeneration there is usually little or no distention, however.

The nerve ganglia, as noted in the section on pathology, may show but an increase of the periganglionic fluid or there may be a degeneration of the cell protoplasm and even of the nuclei. This is the state in the cases of more extensive tympany.

Kader in his experiments found that vascular disturbance, particularly venous hyperemia, was the essential factor in the production of distention in intestinal occlusion. For instance ligation



of a gut was attended by less distention above the occlusion than another part of the gut not obstructed but which had its venous return cut off. It is possible therefore that circulatory disturbances may play a part in the distention of peritonitis. The edema which followed the venous occlusion in Kader's experiments may be absent in loops at a distance from the infection in distention from perforation peritonitis. Schweninger weighed a segment of the affected gut and compared this weight with an equal segment of unaffected gut in order to determine the degree of edema. He found that the weight of a segment may be increased fourfold in obstruction.

Late in peritonitis, distention may be influenced by the edema present but extreme distention may be present without either edema or vascular disturbance.

With more profoundly affected walls and more extensive degeneration there is less distention, the elasticity of the tissues evidently being destroyed by the degenerative process. At least such guts can not be artificially distended.

The degree of distention present may vary greatly. It is determined by several factors. When there is actual obstruction it is naturally great. Distention in such instances seems to be purposive in that by so doing it attempts to widen its lumen. At least there is no sign of a degenerative process. Early distention often takes place over the whole abdomen when only a part of the gut wall is involved and in such areas structural changes can not be invoked to explain it.

I have repeatedly noted that after sewing a window into the abdominal wall the intestines distend and become motionless in the absence of any infection. Distended loops of guts place themselves about the window. That such a maneuver would be a most effective one in limiting infection there can be no doubt, and when infection is added under these conditions the distended loops quickly form adhesions about it if within their power to do so.

This observation causes me to question whether or not in those cases in which nerve and muscle degeneration is present, as above noted, the degenerative changes precede or follow the distention. Experimental evidence leads me to believe that distention precedes and has a purpose just as vascular dilatation and rise of

temperature has, and consequently may be salutary expressions in the course of the disease and not things to be combated merely because it exists.

The motive power for the expansion of the gut is furnished by gases found in the intestine.

That the state of the intestinal contents at the time of the beginning of the disease has much to do with its degree seems unlikely.

Bokai experimented by injecting various kinds of gases into the lumen of the gut. Nitrogen had no effect; carbon dioxide, methane, and hydrogen sulphide produce paralysis while oxygen causes intestinal movements to increase. Paralysis may follow prolonged distention in which instance it is probably due to exhaustion of the muscle or it may be primarily due to reflex involvement of the nerves. There seems to be no evidence available regarding the composition of intestinal gases and their effect on a normal gut. My efforts in this line were expended by conducting gases from an animal affected with distention from peritonitis to the gut of a normal animal. The gut of the recipient distended of course but the effect on the gut wall if any could not be determined.

Late distention is obviously sometimes paralytic. With or without degeneration of the elastic tissue the distention of the gut exceeds the normal range of elasticity of the elastic tissue. In these extreme degrees the elastic tissue refuses the specific dyes. In these late cases there are often degenerative changes in other organs of the body and the changes in the gut wall may be but an expression of a generalized bacteremia. This condition is noted more particularly in puerperal sepsis, a fact that lends color to this hypothesis. The cause of this degeneration must be the toxicity of the exudate, a lessening of the alkalinity, in some cases even an actual acid reaction of the tissue may be noted.

In extreme degrees the distention causes embarrassment of respiration by pressure on the diaphragm. So extreme may be the pressure that cyanosis and dyspnea grow apace with the distending gut. Hypostatic pneumonia and myocardial degeneration may take part in the dyspnea but to deny that pressure on the diaphragm has anything to do with the dyspnea as Van Sweringen does, because a much less lung space is tolerated in tuberculosis, ignores

in part the truth because suddenness in the limitation of air space is not taken into account.

**Temperature.**—In hyperacute cases, particularly those in children, attended by a chill, the initial temperature may be high, to  $105^{\circ}$  or even more. It soon descends. The characteristic temperature is one of moderate height subject to many variations. When a large perforation initiates the peritonitis a primary fall in the temperature to below normal may take place only to rise as the disease becomes established. Not infrequently there is a terminal rise of temperature, sometimes excessively high. I have seen it exceed  $107^{\circ}$  a few hours before death. The characteristic temperature in the acute forms ranges between  $100^{\circ}$  and  $103^{\circ}$ . When the affection tends to localize, the temperature is more apt to hover around the latter rather than the former figure, only to approach normal as the encapsulation becomes increasingly more perfect.

It is not uncommon to find a temperature at or near normal in a very extensive process. As the skin cools with impending death the temperature usually rises. Rectal measurement is the only means of securing accurate determination. The axilla, as noted by Lennander, is altogether untrustworthy and oral measurement, because of the frequent demands for drink or ice, may be very unreliable.

**Circulation.**—With the advent of peritoneal irritation the entire circulation is quickened, expressed at first in a fuller rather than a rapid pulse. As the disease progresses, the pulse becomes more and more rapid. This is quite uniform and is a most reliable sign. It was called the abdominal pulse by the older writers. A rate of 120 to 140 is the ordinary, with an approach to the limit of countability as the disease grows progressively worse. In quality it is often full and bounding in the beginning but the characteristic peritonitis pulse, particularly in the late stages, is small, hard, and rapid. The cause of the rapidity is, in some cases at least, due to irritation of the vagi, reflexly at least at first. Later in some instances there may be directly a nerve involvement. Later there is myocardial degeneration.

**The Exudate.**—In all cases of diffuse peritonitis there is more or less exudate. Occasionally, particularly in those running a slower course, the exudate is confined to a diphtheria-like mem-

brane on the surface of the gut. Often the fluid is sufficient in amount to be readily demonstrable by physical means. The amount of fluid present gives little clue as to the outcome of the disease, but its character may be exceedingly significant.

The physical character of the exudate is variable. In acute progressive cases it is pale milky, and in more localized processes the puriform character is more marked. The odor may declare the kind of organism present. The contained elements consist of leucocytes in more or less imperfect state of preservation. In the most acute cases the formed elements are composed largely of granular debris. In the less acute cases polynuclear and endothelioid cells occur in great abundance.

**General Habitus.**—Early in the disease the features may be expressive of acute pain even to the presence of cold perspiration. The patient is apprehensive lest the surgeon's manipulations will increase his pain. As the disease subsides the expression of the patient may exhibit tranquility. It is the sign of localized inflammation. If the disease progresses, the face may become flushed and there is apt to be a yellowish or grayish tint of the skin about the alæ of the nose. The eyes are wide but tend to be sunken. The patient despite his attitude of resignation is apt to move his limbs restlessly about while maintaining an immobile trunk. He often inquires as to when he shall be given relief. In this stage the life of the patient hangs in the balance. It is a sign of advancing inflammation.

When the disease advances toward a fatal termination the characteristic phenomena are pinched features, sunken eyes, pale skin, white closely drawn lips, a sharp pinched nose, and above all a glaring, glassy eye. Delirium may take place in the terminal stages but the mentality is often hyperacute, the demeanor hopeful or indifferent. I recall the case of a young man in these final stages who lay and discussed the prospects of the fall quail shooting as his extremities gradually cooled in death. I recall a young man who greeted my approach to his bedside with the remark that I had saved his wife, and I should now save him and he added "but you'll have to hurry." His further conversation indicated that he had anticipated an operation in anxious hope.

I have often wondered as to the mental content in these cases.

They lie apparently alert, yet usually uncommunicative, taking little notice of their environment. Anxiety has given way to apathy. I believe that the nerve cells are so affected by the circulating toxins that they are incapable of producing emotions of hope or fear. The glare of the eyes most likely does not indicate alertness as we are wont to assume in the final stages. The shrunken features retract from the eyes giving them an undue prominence. They no longer follow the movements of those about them. The pale skin and shrunken features but bespeak the condition of the extremities. The peculiar cold clammy feel is not imitated in any other condition and most closely resembles the peculiar cold feel of a dog's nose.

With the cooling limbs, the eyes become more and more sunken, the features more drawn. The ascending temperature fails to change the cutaneous circulation. The patient responds to questions and we say he is conscious, but he is indifferent to his environment. His children do not interest him, the surest sign of departed comprehension. Of all the environment he suffers the least.

In some instances an orientation with his surroundings seems to be retained to very near the last. This is true in those cases where there is a paralytic ileus which dominates the field, or some other condition which hastens the final end, before intoxication has had time to veil his comprehension.

**Physical Characters of the Abdomen.**—Examination of the physical characters of the abdomen may reveal much. The scaphoid abdomen and its opposite, extreme tympany, tell the most. The one feature in common is immobility. The excursions of respiration are notably absent. In localized processes only a part of the abdomen is immobile. When an appendicitis has become localized the left half of the abdomen may not be fixed and in pelvic peritonitis it is quite common for the upper abdomen to share the respiratory excursions. Sometimes certain areas may show distention not shown in equal degree by contralateral regions. This is particularly valuable in children. A localized abscess or an agglutinated mass of intestines may produce a bulging of the abdominal wall. A localized abscess containing gas-producing bacilli may show distention beyond the unaffected side. When such a

state is attended by hypertympany it is a sign of great value. The bulging of the navel may occur in children even when there is no fluid. Vascular dilatation may be noted when tympany obscures a paraperitonitis localized in one point. When in a child uniform distention and tympany is attended by venous hyperemia it may be very suggestive of local reaction.

The hand of the examiner usually follows his eye in the elicitation of the phenomena and assists in their interpretation. Local resistance in spite of tympany may indicate a reaction beneath. The abdominal wall over a peritonitic area may suggest the presence of a neoplasm. The presence of a localized abscess or omental tumor is one of the most common signs. Battle noted edema of the abdominal wall in the region of the anterior superior spine in several cases of peritonitis. It is common enough to observe edema of the deeper layers of the abdominal wall during the course of an operation, but its existence to a degree sufficient to cause pitting must be unusual. I have seen this phenomena only very late when a long neglected abscess presented beneath.

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## CHAPTER XIV

### DIAGNOSIS OF PERITONITIS

The simple abstract question as to the presence or absence of peritonitis often perplexes the examiner. If peritonitis is present the question whether it is irritative or suppurative needs to be decided before treatment can be formulated. There are no signs taken alone which are pathognomonic of peritonitis and it is only the association of several of these and particularly as to their sequence and manner of onset that is significant. There seem to be no fundamental facts in the minds of many practitioners as to the relative importance of various signs. It seems worth while to attempt to formulate general rules, based on my own errors and those of others.

The general phenomena attending peritonitis have been enumerated in the chapter on symptomatology. The attempt here will be to call attention to signs which may be observed in peritonitis but which may likewise be observed in conditions not attended by inflammation of the peritoneum.

The cardinal symptoms of peritonitis are pain and local reaction. These bring with them muscular rigidity and constitutional reaction. The statement is sometimes made that peritonitis may exist without the presence of pain or fever. That is true, but it is not true that peritonitis can exist without constitutional disturbance. The thermometer is not the only measure of reaction. The eye of the practitioner must supplement the thermometer just as the sense of touch must check up the pulse rate as shown by the chart. Temperature of  $98.6^{\circ}$  and a pulse rate of 75 when recorded on the chart may indicate a normal patient but a look at the patient and the feel of the pulse may indicate that grave things are pending. In order that the practitioner shall answer the question of the presence or absence of peritonitis he must be able to evaluate the symptoms by their presence or absence.

In order to analyze the various symptoms common in peritonitis

it will be well to enumerate them and discuss their direct and differentiating value in determining the question of the presence or absence of peritonitis in the concrete case.

**Pain.**—This is the most constant sign in peritonitis. Abdominal pain is also present in many other abdominal conditions. Diseases that antedate the peritonitis may be caused by other factors. The characteristic of the pain in peritonitis is that it is increased on movement. It is a friction pain, and anything that increases the rubbing together of the inflamed surfaces on each other increases it. In this regard peritonitis closely parallels pleurisy and much can be learned by watching the protective efforts of the patient in this disease. The factors which increase the pain may be either the movement of some part of the body, as the iliopsoas in appendicitis when the thigh is lifted, the movements of the diaphragm in respiration, the emptying and filling of the rectum, etc. In pleurisy the necessary movements of respiration make it possible to note the influence of movement on pain. In the case of the peritoneum these movements may be wanting and the surgeon imitates these movements by manual pressure in the region of the supposed inflammation. Two axioms may be laid down for the pain in peritonitis: (1) The pain produced by pressure is proportional to the pressure or the range of the voluntary movement incited to produce the pain. Pains relieved by pressure are not due to inflammation. In neurotic conditions superficial pressure may cause pain, while deep pressure causes none. (2) The pain of peritonitis is localized at the site of the inflammation. Pains elsewhere are due to reflex or referred pains or to hyperemia or edema. Visceral edema may cause referred pain as in cholecystitis and appendicitis, but these are splanchnic pains and have nothing to do with peritonitis, though they are often followed by it. In violent inflammations the area of pain may be widespread, because the area of hyperemia and edema may spread beyond the actual site of infection. When spontaneous pain and pain on pressure coexist, the limits of each must be determined. For instance in the sudden perforation of an organ there may be diffuse pain due to reaction of the sympathetic nervous system while the pain due to actual inflammation may be much more limited. The statement of the patient may be taken to determine the referred pain while manual palpation will secure



information as to the limits of inflammation. In such instances the tenderness is the guide to the degree, and not the area of spontaneous pain. Patients may complain of diffuse pain and the whole abdomen may be rigid, but some area of this rigid abdomen shows the greater sensitiveness. This area of tenderness is the guide to the extent of the involvement. For instance, in a perforated duodenal ulcer, diffuse pain may be complained of and the whole abdomen may be rigid, but the site of greatest tenderness will be over the duodenum and over the ascending colon.

The site of the initial peritoneal pain is significant. It is where the infection starts, not necessarily the site of the source of infection. The site of the initial peritoneal pain is not parallel with the initial pain of the disease. For instance appendicitis is often initiated by an epigastric or diffuse pain. This pain is diffuse, not associated with local tenderness and is due to a splanchnic nerve irritation within the walls of the appendix. When the infection escapes from the appendix the peritoneum is irritated and the local pain begins.

Pain in the abdomen may be the result of a variety of conditions not associated with inflammation of the peritoneum. The most common of these may be enumerated.

**Colic.**—Violent contractions of a hollow viscus may cause pain. The more common of these are due to the attempts at the propulsion of a foreign body, possibly in part by the irritation produced by the foreign body itself. Familiar examples are renal, gall bladder and intestinal colics due to the contraction of their walls. The essential feature of these is sudden onset of great intensity, without the signs of local reaction. The patient presses over the painful area, rolls about and in general is indifferent to his position. There is no rise in temperature and the pulse is responsive only when the pain is on. Infection may be associated with the colic and then there may be fever and leucocytosis, and a measure of tenderness when a diseased wall can be directly pressed upon. It is only when the peritoneum is reached by the infection that the pain on movement appears.

**Thrombosis.**—When an organ becomes necrotic from obstruction of its blood vessels, pain is produced. The pain in these cases is due to the nerve irritation due to the dying tissues. This irrita-

tion may be due to chemical irritation, in part to the stretching from the extravasation that always accompanies these conditions. Gangrenous appendices, mesenteric thromboses, and cysts with twisted pedicles are familiar examples. These conditions are attended by sequelæ. Following the initial pains comes the secondary pain of irritation. The necrotic organ is an irritant to the surrounding peritoneum and a violent peritonitis is set up. This peritonitis is plastic in character, prone to form adhesions as is commonly seen about ovarian cysts, and is distinctly consecutive in character inasmuch as it is able to give temporary nutrition to an injured organ. This stage is attended by a considerable exudate, sometimes enough even to admit of demonstration by physical means. Absorption of these exudates may produce temporary leucocytosis and rapid pulse. Necrosis of the organ alone is not attended by local tenderness, but when the reactive processes begin all the signs of peritoneal inflammation appear. Perhaps this is well, for, save in the case of the cysts with twisted pedicles, death of the wall is apt to take place, followed by perforation and a general septic peritonitis.

Probably the severe abdominal pain due to pancreatic necrosis belongs here. Pain due to tearing of tissues, if there be hemorrhage, may be added. The distention of the guts and the rapid heart are likely caused by the close proximity of the lesion to the large ganglia. Exudate follows the initial lesion and peritoneal irritation is produced.

**Intraperitoneal Hemorrhage.**—Coagulated blood acts as a chemical irritant. This may be frequently observed in the blood clot from tubal abortion, hematoma of the ovary, etc. The fibrin of the blood clot excites exudation in the surrounding peritoneum and with this comes pain. The normal course of a blood clot pain is short, soon reaches its height, and gradually recedes. The pain is due virtually to a chemical peritonitis. Blood not coagulated does not produce pain. In cases therefore in which there is extravasation of blood the pain from the clot must be differentiated from the pain attending the lesion from which the bleeding results, for instance the cutting, tearing pain of tubal abortion is very different from the pain of blood-clot irritation. The pain due to the presence of a blood clot produces a pain of moderate intensity and is equal

to the pain produced by an infective process in the stage of fibrin formation—less acute than in the primary infective processes. The pain of a blood clot in the pelvis may be compared to the acute pain of peritubal inflammation. In determining the different causes the time element must be considered. A blood clot produces the kind of pain in a day which attends peritubal infection after a week. The same principles apply in any other region.

**Pain Caused by Distention of Parenchymatous Organs.**—When an organ becomes suddenly distended pain is produced. Acute hyperemia of the kidney due to multiple thrombi, septic or aseptic, is attended by severe pain. The acutely distended liver from sudden failure of cardiac compensation may simulate an abdominal infection very closely. Infective processes in the liver are seldom large enough proportionate to the size of the organ to produce a distention pain of any magnitude, and the kidneys are seldom markedly painful in heart failure. The pain within the abdomen by virtue of heart failure may entirely overshadow the heart lesion. The rapid heart may erroneously be ascribed to an intraabdominal infection. The big liver, and the diffuse apex beat which is nearly always displaced outward should place the surgeon on his guard. The lack of tenderness is distinctive.

**Referred Pains.**—The thoracic and abdominal walls serve as conduits for the same nerves and it can not be wondered at that irritation in one part produces sensations experienced in other regions. It is comparable to "listening in" on party telephone lines. A pleural pain, it may be readily understood, may irritate the intercostal nerves and the sensation be transmitted to the abdominal wall. The pain may be felt in the latter place and not be felt at all in the pleura. There may be an absence of physical signs indicative of pleural disease, particularly in children. Here rapid respiration may be the first clue and a flaccid abdomen may give further proof. When there is referred pain from the pleura the abdominal wall may be rigid and painful to touch but the pain as produced is greatest on superficial contact and lessens as the pressure is increased, just the reverse from the relation in peritonitis. The muscular rigidity may be marked but at just that moment where inhalation passes to exhalation the tenseness of the muscle lessens as it never does in peritonitis.

**Neuroses.**—Neurotic persons commonly have abdominal tenderness. This is a superficial pain and is dispelled if the patient can be engaged in the fascinating topic of her own ailments. If there is muscular rigidity it applies to the recti alone and not to the oblique muscles. Palpation may cause a violent contraction of the muscles, a movement which would be very painful were a real peritonitis present. Often these neurotics find the examination exceedingly funny and respond with a giggle. There is no fear of peritonitis in such cases.

**Rigidity.**—Rigidity of the abdominal muscles is the most difficult sign to interpret. It may be due to a great variety of conditions not associated with disease of the peritoneum. For instance, rigid abdomens may accompany cerebral irritation, be this due to meningitis or modesty. The various types may be very difficult to differentiate and the practitioner must often call associated conditions to his aid. Rigidity of central origin, whether due to inflammation or mental obfuscation, is accompanied by fixed retraction. The rigidity due to sensitiveness is spasmodic and is accompanied by contraction of the adductors of the thighs and often of the pectoralis and biceps. These may be described as biological defensive movements. The diagnosis of a cerebral disease or of nervous hypersensibility does not end the problem, for a typhoid patient or the overmodest maid may each suffer from a peritonitis.

Once the reactive process is defined the rigidity of peritonitis involves those segments of muscles only which cover the area of inflammation. The sharpness of the border of the rigidity is dependent on the degree of limitation of the inflammatory process. In acute inflammation where the hyperemia is diffuse the rigidity is usually more extensive than the actual site of infection, as already discussed, while when the process becomes limited the rigidity may become so sharply circumscribed that a tumor is simulated. In fact the peritonitis may wholly subside and but an encapsulated mass remain.

Sometimes the peritoneal covering of an organ may be inflamed and yet no muscle rigidity may be present. The inflamed area of peritoneum may not be accessible to pressure and muscular rigidity is absent. This is observed in lighter degrees of cholecystitis, sal-

pingitis, or appendicitis when that organ hangs deeply in the pelvis. In such instances we bring voluntary movements to our aid, or seek by other means to subject the diseased organ to pressure, or observe the effect of the movement of neighboring organs. For instance in inflamed gall bladders we press deeply over this organ and ask the patient to breathe deeply; in salpingitis vaginal examination is made to aid abdominal palpation; and the effect of the filling and emptying of the bladder and rectum on an inflamed appendix situated in the pelvis is noted particularly in children.

**Fever.**—Fever is of little value in making a diagnosis of peritonitis. It is usually present, particularly in the beginning, and its absence if measured consistently in the beginning of the attack is a presumptive sign against it, though no doubt the peritoneum may be inflamed when no fever is excited. The value of this sign in peritonitis is lessened because there are so many diseases accompanied by fever.

The fever characteristic of peritonitis is not high. A temperature of  $99.5^{\circ}$  to  $103^{\circ}$  usually represents the limits of the temperature curve. If there is high fever the presumption is against the presence of peritonitis. High fever may come later when there is a walled off abscess, but high fever in the beginning is seldom observed. The temperature may be subnormal. This indicates not a reaction but an intoxication, and is not a measure of reaction but of suppression of reactive forces. It is the severe case that is accompanied by a subnormal temperature. The period of the rise of temperature is not coextensive with the inflammatory reaction.

Fever often subsides long before the reactive process ceases. Fever is a measure of reaction against toxins absorbed and when absorption ceases the fever subsides.

**The Pulse Rate.**—The pulse rate, like the fever, is a measure of general absorption and only in a general way runs parallel with the local reaction. It is sometimes said that peritonitis may exist in the presence of a normal pulse. This is not true. A pulse may beat at the normal rate per minute yet be far from normal. Peritonitis is not incompatible with a slow pulse, but there are changes in character that will not escape the careful observer. It may be slow and quick, full, semidicrotic, etc. The characteristic pulse of

peritonitis is a rapid one in proportion to the temperature. A slow-ing pulse usually accompanies a receding area of tenderness.

**Leucocytosis.**—There is an increase in the number of leucocytes in the blood whenever there is a considerable degree of reaction on the part of the peritoneum. A proportionate increase in the polynuclear leucocytes is particularly significant. This sign is of value when other diseases of the abdomen are present which are accompanied by fever and intestinal disturbance, notably typhoid fever. Generally speaking, the number of leucocytes runs parallel with the extent of the infection, and is particularly likely to run high when abscess formation begins. On the other hand the white count may be below normal. This leucopenia is present when there is a general intoxication, whether there is a peritonitis present or not. Subsidence of leucocytosis is not a sign of a recession of the process but of the absence of absorption of toxins. This may take place when the abscess becomes encapsulated. If the capsule becomes broken by the surgeon or from increasing tension the infection may spread again. The greatest delusion connected with leucocytosis is that a recession necessarily indicates an absence of infectivity.

**Tympany.**—Abdominal distention is a frequent accompaniment of peritonitis, but is not a positive sign of *appendicitis*. At first it is reflex in character and is purposive. Later it is due to direct irritation of the infective material and serves the very valuable purpose of exposing the greatest possible extent of surface toward walling in the infection. It may later become paralytic and become the most serious complication. Tympany without local signs of reaction is not a sign of peritonitis. On the other hand many cases of tympany are associated with pain. These may be due to reflex disturbances or to actual occlusion of the gut to such an extent that fecal circulation is interfered with.

Intestinal obstruction is characterized by distention as an early prominent symptom. There is lacking the localized tenderness and rigidity, fever, and leucocytosis. Local pain may cause peritonitis to be simulated if the cause of the obstruction is such that a reaction is produced. In some cases a tumor may be produced, as in intussusception, but there is no muscular rigidity surrounding it. Vomiting is common in acute obstruction, but it comes on

after the distention develops, while in peritonitis vomiting is early and if tympany occurs it follows the vomiting. Late vomiting may occur in peritonitis, due to mechanical or dynamic ileus. At this stage the diagnosis can not be in doubt.

Severe contusions of the spine may be attended by pronounced tympany. These usually begin early, sooner than tympany would occur were there a peritonitis. This sign is distressing to the observer for it may mask a peritonitis which may be set up by an associated lesion, such as a traumatic rupture. The tympany may be so great that the pulse rate may be increased, but there is no tenderness or fever.

## CHAPTER XV

### PROGNOSIS OF PERITONITIS

The outcome of any given case of peritonitis is dependent on a great number of factors. The cause in itself may be a determining factor. If an ulcer ruptures or the gut wall is injured so that there is a free escape of gut contents, the patient will most surely die. Here the dual elements of bacteria and foreign body reach the unprotected peritoneum in amounts sufficient to overwhelm the defensive forces. When there is not a complete perforation and bacteria alone escape, then the problem becomes more complicated for the type of bacteria that have escaped and the resistance the body is offering can be determined in a general way only from the symptoms or possibly from fluid obtained at the time of operation. In the first group of cases, the perforative, statistics are able to give reliable data as to the influence of operation. In the group in which gradual escape of infection has taken place statistics are quite valueless since classification is wholly a matter of personal equation.

It may be profitable to view a few statistics of the influence of time of operation on the cause of the acutely perforative cases. The results obtained from the examination of fluid removed at the time of operation have as yet given no reliable data, but the possibilities of the future warrant the keeping of this line of research in mind. When the surgeon is confronted with the problem of prognosticating the outcome of a concrete case he must rely on the course the disease has taken to date and the symptoms manifest at the time the summary is taken. The more prominent of these guides may be detailed with the hope that they may be of some use to the beginner.

**Prognosis Dependent on Time of Operation in Perforating Ulcer.**—Miles (Observations on Perforating Gastric and Duodenal Ulcers Based on a Personal Experience of Forty-six Cases Operated on, *Edinburgh Med. Jour.*, 1906): Cases operated on in the



first twelve hours give a mortality of 26 per cent, those operated on between twelve and twenty-four hours give 45 per cent mortality and 92 per cent in those operated on thirty-six or more hours after perforation.

Fenwick's statistics (*Ulcer of the Stomach and Duodenum*, P. Blakiston's Son & Co., Philadelphia, 1900) are as follows: Those operated on in from one to twelve hours, 33 per cent; twelve to twenty-four hours, 50 per cent and more than twenty-four hours, 86 per cent.

These statistics give a general idea of the importance of prompt treatment, but none as to the outcome of a given case.

**Prognostic Value of Examination of Peritoneal Fluid.**—The number and virulence of the bacteria in an exudate can be determined in a general way by the examination of a smear at the time of the operation. The number can be estimated per field for usually early in the course of the disease the bacteria in the field are within the range of mathematical estimation. Many extensive exudates are nearly bacteria-free. This is particularly true of the odorless slightly floccular exudates. By constant examination one learns not facts but general impressions. The virulence of an infection may be determined in a general way by the action of the endothelial and polynuclear leucocytes. When the bacteria are largely or wholly extracellular and particularly if the leucocytes show evidence of disintegration the infection is a severe one. If the leucocytes have englobed the bacteria, the infection is a mild one. The time element must be considered. Early in mild infections the bacteria may be extracellular. The later the stage the bacteria are found extracellular, the graver the prognosis.

**Prognosis According to Species of Organism.**—In most cases the type of bacterium predominating can not be made out from a slide examination. In some instances where the source of the infection is known this may be a valuable clue. If a secondary streptococcic abscess has been allowed to contaminate the general peritoneal cavity, the prognosis is very grave. Even in slide examinations streptococci may be made out and when they dominate the outlook is grave. The presence of *B. pyocyaneus* can not be determined by the preliminary examination, but when identified by

culture or the appearance of blue green pus it indicates that the course will be a very long and stormy one.

The foregoing remarks offer but little aid to the experienced surgeon, valuable as they may be to the beginner. To the clinician the general appearance of the patient is intuitively interpreted. The result of this intuition is usually that he is very guarded in his opinion and leaves a wide leeway for subsequent developments.

From what has been said in the chapter on pathogenesis and pathology it is apparent that the discussion of the symptomatology as an element in prognosis lends itself poorly to abstract consideration. A careful observance of all the phenomena gives certain definite clues as to what may be anticipated. For instance, by the mode of onset of peritonitis associated with an appendicitis it can be judged whether periappendiceal adhesions have formed or not. A proper appreciation of such phenomena can be obtained only by observing clinical and pathologic material. Certain signs may be collected here, however, that may be of some service, as a chart to the juvenile mariner sailing for the first time on the troubled sea of abdominal surgery.

It must be appreciated that the fundamental problem in prognosis resolves itself very largely into judging whether or not the process will spread, and if so how much. The anatomic conditions under which spreading takes place have been discussed. When all the data are at hand this admits of fairly accurate consideration. Early in the course of the disease, however, when only a portion of the data may be at hand, it may be extremely difficult to determine this point, sometimes even after the abdomen has been opened. If one can observe the patient for a period of some days, or if clinical data have been recorded, one can determine how far the disease has spread during that time and determine from this what the effect will be on the welfare of the patient. To judge how far an incipient disease will spread when observed within the first hours, admits of judgment only within the widest ranges. It is under such circumstances as these that statistical data are of value.

When circumstances are such that no definite knowledge can be obtained as to the pathologic physiology, either because of the nature of the case or because of the limited experience of the ob-

server certain data can be obtained by the statement of the patient and by the general state of bodily reactions. The various cardinal symptoms may be reviewed with this point of view in mind.

**Pain.**—The *initial pain* gives some idea as to the degree of severity of the inflammatory process. A mild appendicitis may present itself as a soreness in the region of the appendix, with but little general disturbance. A perforation of a duodenal ulcer on the other hand begins with the most intense pain, pronounced general disturbance, and early general abdominal tenderness and rigidity. When the pain is slight and subsides early a mild lesion is indicated, provided that no anodyne has been given. When an anodyne has been given this sign is confused. Even when morphine has been given the effect a single dose produces may be of some aid in judging the intensity of the pain. If the patient goes into a prolonged sleep on a sixth of a grain, one would hardly suspect a perforating ulcer. Pain is the product of the reactive inflammation and in those cases which are predominantly toxic, pain loses its prognostic value. This is well marked late in the disease in general peritonitis when euphonasia supplants suffering.

Ordinarily the pain of a well-localized lesion subsides in three or four days. The diffuse pain lessens and only the immediate site of the lesion is painful. The salutary cessation of pain from peritonitis is gradual. Spontaneous pain first lessens, then pain on movement and finally pain on pressure.

When the inflammation spreads there is an extension of the painful area, as is often seen when an appendiceal peritonitis spreads across the pelvis to the left groin. The same is true when the sub-diaphragmatic region becomes involved. Local pain is prolonged if the reaction extends to a suppurative stage. When active abscess formation begins renewed pain from distention of the tissue from the increasing size of the abscess results. This increase in pain must be distinguished from renewed pain from a spreading of the inflammation.

A sudden cessation of pain on the other hand may be of the gravest omen. Distinction here must be made between a spasm pain, pain from the inflammatory process itself, and pain from the mechanical distention of the tissue. Spasm of the gall bladder may relax and the patient feels as well as ever within a very short time.

A pericholecystitis usually requires several days before any degree of comfort is reached. The rupture of a distended gall bladder may give sudden temporary relief. The antecedent factors here must be the guide. When a gall bladder ruptures the surcease from pain does not bring the calm quietude of restored health. It is rather an ecstatic state, a sort of ensthanesia, a salve of nature to fortify against the trouble yet to come.

Sudden cessation of pain, after a considerable degree of reaction pointing to abscess formation, spells disaster. Sudden cessation of pain in a patient who has fever, increased pulse rate, and leucocytosis means a spreading, and nearly always a spreading in an unprepared field from which limitation without operative aid is not to be anticipated. When an abscess breaks into the intestinal tract the results may be salutary. Here the temperature and pulse approach the normal quickly after the cessation of pain.

**Fever.**—The initial fever in peritonitis is not high. A localized peritonitis, if it is subsiding, should show a reduced temperature at least by the third day. If it continues to ascend beyond this period either the peritonitis is spreading or a localized suppurative process is in progress. If the temperature suddenly drops an accumulation has ruptured, either within the free peritoneal cavity or elsewhere. If in the free peritoneal cavity it drops to subnormal and the pulse mounts, if elsewhere as an appendiceal abscess into a gut, the temperature does not become subnormal, and the pulse rate also drops. When fever and pulse rate part company, disaster is impending. This rule applies to all diseases.

**The Pulse Rate.**—The rapidity of the pulse rate is dependent on the general systemic reaction as measured by the suddenness of the onset and the degree of toxicity. It is the most valuable single prognostic sign. An increasing pulse rate implies an extension of the process and the anxiety of the surgeon can not abate so long as the pulse rate continues to mount, despite any sign of improvement of other symptoms. As a matter of fact an ascending pulse rate is made more grave if in the face of this the patient progresses toward a state of well being. If with an increased rate the volume becomes smaller and more tense impending disaster must be recognized.

**Leucocytosis.**—Very generally speaking leucocytosis bears a re-

lation to the state of the inflammatory process. When the infection is severe there may be a leucopenia. An experienced clinician would hardly be fooled by a "normal" count, since infective processes severe enough to repel leucocytes bear unmistakable earmarks of gravity. Leucocytosis may fall as localized suppuration develops, since the white count is an index of the toxin's reaching the blood stream and not of what may be pent up somewhere in a walled-off abscess. It is no evidence that the process is subsiding without suppuration when a localizing process is attended by a subsiding leucocytosis. If the white count begins to mount when an abscess is known to exist even without increased pain, a spreading of the infection is likely. This may be seen sometimes in a walled-off appendiceal abscess. Spreading may take place toward the pelvis without renewed pain but with an increased leucocytosis. This is usually true in those cases in which agglutinating inflammation has extended beyond the site of an abscess. When no such agglutination has taken place the spreading infection causes renewed pain corresponding to the area involved. Sometimes when the ruptured abscess is excessively toxic renewed leucocytosis and pain may be prevented by the extreme toxic absorption. The patient may pass from the stage of freedom of pain caused by relief of tension of the abscess, to the euphonasia of extreme toxemia. I recall a man who had been writhing with pain from a periappendiceal abscess when the cot on which he lay broke, precipitating him to the floor. Pain ceased at once, but the leucocytosis doubled. Despite warning he refused operation until pain should indicate a renewed inflammation. No pain appeared and a diffuse peritonitis was revealed by the autopsy.

On the whole leucocyte determinations must be made at regular intervals to be of use. To the young surgeon the information it gives is invaluable but the experienced surgeon becomes independent of the information it gives.

**Muscular Rigidity.**—When the infection is becoming localized the muscular rigidity gradually subsides. Thus a rupture of an abscess into the peritoneal cavity may bring relief from pain but the muscles become tense even in the diffuse toxic type. This is a danger signal of the greatest importance.

**Tympany.**—Initial tympany may be expressive of a reflex aid

to the walling in of the infection. Increasing tympany after the process has once become established is of the greatest gravity. Tympany is never so grave as retraction. A scaphoid abdomen usually indicates a grave state. If attended by a leaky skin and rapid pulse it means a wide extension of the exudate, often without reaction on the part of the peritoneum, and consequently a speedy, fatal termination.

**Sordes.**—A dry, brown furred tongue is a grave omen and when associated with euphonasia spells impending dissolution. A widely spread inflammation in the presence of a reaction may show a dry tongue and yet recover. For this to come about there must be a leucocytosis, some temperature and a hot skin. When a dry tongue begins to moisten it is a favorable sign.

**Singultus.**—Hiccough is a grave sign. It means irritation of the diaphragm, either from pressure from distended guts, or the extension of the infection to the diaphragm itself. If due to distension recovery may follow; when due to spreading infection death always follows.

**Vomiting.**—Vomiting in the beginning is common and is then due to reflex irritation through the sympathetic. Late vomiting is due to obstructive or paralytic ileus and spells early dissolution. In rare instances an obstructive ileus may recover.

**Complications.**—The advent of associated lesions always adds gravity. An infective pleurisy, lung abscess, venous thrombosis, cerebral disturbance, joint infections, all add their quota to the burden the patient has to bear. This influence must be evaluated in each instance. Usually it may be said that when these complications appear the peritonitis has subsided beyond the danger point. The prognosis then becomes the prognosis of the complication. The factor of importance is to recognize the changed or added phenomena as complications and not regard them as variations in the symptoms of the primary disease.

## CHAPTER XVI

### CAUSE OF DEATH IN PERITONITIS

In no chapter in the study of the peritoneum is the state of our knowledge so unsatisfactory as that concerned with the cause of death in peritonitis. Perhaps it may be admitted that, broadly speaking, any fatal disease becomes so either through a failure of respiration or of the circulation. Be this as it may, the avenues which lead up to either of such catastrophies remain unexplained in the case of appendicitis. Obviously enough knowledge of the sequential development of deleterious phenomena would be of vast importance in the formulation of a scheme of treatment if we possessed it. However, we have no such knowledge and once the disease has passed the stage of its focal origin the surgeon is without a fundamental scientific basis for subsequent procedure.

Generally speaking, two main theories have been advanced to explain the cause of death in peritonitis: that it is due to a septic intoxication, and that a shock to the nervous system is the cause of death.

**Septic Theory.**—This theory assumes that death from peritonitis is due to the absorption of some sort of poison from the inflamed peritoneum into the circulation whence it is carried to some vital organ, there to exert its baneful influence. Wegner was the first to emphasize the claims of this conception. Studying the results of infection after abdominal operation, in consideration of the wide extent of the peritoneum and its capability of absorption, he concludes that in these factors must be sought the explanation. He bases his strongest argument on the fact that death may occur before local changes have taken place. Krönlein and Bumm supported this theory and Reichel also accepted it with qualifications, namely, that it be applied to the acute types while the localized pus-producing varieties presented additional factors which will be enumerated below. Fraenkel and Kraft held the same

views. Körte and Strümpell support Wegner in the main but admit the possibility of some reflex influence.

The main support for the septicemic theory is found in the constant presence of a bacteriemia in this disease. Barbacci maintained that the bacteria appeared only in the agonal stages of the disease. This contention is no longer possible since bacteria are now with perfected technic regularly recovered from the blood stream in the earlier stages, and from the parenchymatous organs in experimental peritonitis in animals even in the earliest stages of the disease. Tietze was one of the first to emphasize this point, and Waterhouse also emphasized it.

**Nerve Theory.**—The basis for this theory is the observation of Goltz that tapping the splanchnic area in frogs regularly produces death. A full consideration of the possibilities of this theory would involve a consideration of the theories of shock. The chief modern contender for this theory was Ziegler. His chief reason apparently for supporting it was the failure to secure positive blood cultures. According to him death is due to reflex irritation due to the action of bacteria on the peritoneum.

A number of unclassified theories have been advanced which may be recorded with the hope that their further development may bear fruitful results. Grawitz believed that it was the extensive local suppuration which, by abstracting albumin from the circulation and by the high fever consequent to it, produced a parenchymatous degeneration of parenchymatous organs, notably the liver and kidneys. Bauer believed it was the abstraction of water from the tissues because of exudation and vomiting which exerted the bad effects. He admitted also the possibility of a reflex influence on the nervous system.

A summary of the evidence is facilitated by considering separately those cases in which death occurs before extensive changes in the peritoneum develop and those where the local changes, suppuration, pseudoileus and the like take place.

In rapidly fatal cases death may ensue within a few hours, possibly even less. This is seen in puerperal infections and after perforation of ulcers. That these may cause death by a species of shock can not be denied. The injection of chemical substances is sometimes rapidly fatal in small amounts. I have experimented



particularly with turpentine, iodine and olive oil. In the first two there is intense irritation of the peritoneum and death may ensue before there is evidence of any injury to the parenchymatous organs. Small amounts of silver nitrate injected into the peritoneum are fatal, even in dose ranges which are well borne when injected subcutaneously. In none of these can the chemical toxicity be excluded. Ice water injected into the free peritoneal cavity may produce death in a few minutes in amounts not fatal when injected intravenously. Death in this instance must be ascribed to unknown influences, popularly called shock. That like results may be caused by acute infections can not be denied or proved.

Nevertheless, in the majority of rapidly fatal cases an absorption of toxins, probably of bacteria themselves, is much more tangible. How this acts is quite another problem. In experimental injections of bacterial cultures in animals no lesion recognizable by the microscope is discoverable. Possibly some potent factor like anaphylaxis is operative. If this be true it is but substituting a word of unknown meaning as a symbol for an unknown process. In some cases small petechial hemorrhages are found in the central nervous system. In less acute cases cloudy swelling of the kidneys and liver is often observed. Generally speaking, it is only in cases which have run a course of forty-eight hours or longer that the above mentioned anatomic changes are noted. It is possible in the light of newer opinion that functional disturbance may precede the organic lesion.

In the less acute cases, which are common ones, which run a course of from 4 to 8 days the series of phenomena is far more complicated. In such cases meteorism, vomiting, pseudo-obstruction from kinking of the gut, are common observations. These serious factors probably play a varying part in the different cases. I have made the problem as to why the intestinal canal dilates a special study in order to determine the cause of the meteorism. There is no obvious change in the nerve plexuses surrounding the gut wall. These nerves and their fibers stain as in the normal gut. The muscular coat of the gut shows definite changes, however. The muscle cells show a fine granular change quite like the earlier changes in cloudy swelling of the kidney. These show in cases running a course of twenty-four hours or more. In the hyperacute cases this

change is not manifest. Whether this again represents a loss of function antedating anatomic change is a matter of speculation.

The injurious effects of such dilatation likewise are not clear. That great mechanical discomfort may ensue is obvious. Whether the associated dyspnea is due to mechanical pressure on the diaphragm, degeneration of the diaphragmatic or other respiratory muscles, or to some action of the nerve centers is difficult to answer from anatomic evidence. Mere increase of intraabdominal tension as by the return of large masses of intestine during operations for large hernias may result in distressing even fatal dyspnea, I have learned by experience. Dyspnea associated with great tympany, even in the most distressing cases, may be lessened temporarily by puncture or drainage of a gut. I am disposed to believe, therefore, that the problem is largely mechanical, despite the fact that degenerative changes may sometimes be noted in the diaphragmatic and intercostal musculature. In fact this degeneration may make the great distention possible.

Recent studies on intestinal obstruction make it seem possible that absorption from the intestinal canal, particularly from its first portion, may exert a deleterious effect. It can be easily demonstrated, however, that when such conditions exist absorption both from the serous and mucous surfaces is very much slowed.

In the study of human material the picture is often much more complex. Generalized cloudy swelling of and hemorrhages into the parenchymatous organs is common. Secondary abscesses may form in an endless number of situations.

That loss of fluid exerts any particular influence as Bauer thought is unlikely, since the amount so lost is really small and may be much exceeded in other diseases which are not fatal.

That the effect of local suppuration is other than salutary, comparatively speaking, is unlikely. The older authors were quite right in their regard for pus. If one must have infection as they did, the development of pus is a laudable event. This fact is as true now as ever. Suppuration merely means that an inflammatory reaction has taken place and when such is the case absorption is markedly lessened. When death follows suppurating processes the deleterious action is lessened in direct proportion to the encapsulation of the mass. Death when it follows such a state is due to the com-

plication of the process by the occurrence of a renewed infection in the immediate vicinity or at some distant point in the form of a metastatic abscess. Then the problem becomes the same as above detailed for the nonsuppurative type.

The problem as to the cause of death apparently centers about the absorption of toxins or bacteria, most likely both, and their action on some vital organ or organs the identity of which is at present unknown. How toxins may affect the organisms when absorbed has been the object of numerous studies. One of the most careful of these is by Heineke. In experiments upon animals he determined that the blood pressure remains near normal until severe constitutional effects had developed. The fall then was gradual. After considerable fall had taken place by increasing the amount of blood in the heart by pressure upon the abdomen or clamping of the aorta the pressure again rose. He concludes that the loss in pressure is due to lessened blood in the heart. This he ascribes to lessened tonus in the vasomotor center. Since these changes are progressive, death must be caused by progressive loss of the tonus of the vasomotor center.

The question has been raised as to why infection of the pleural cavity differs in course and fatality from that of the peritoneum. The answer is simple: it does not. The ordinary pleurisy runs a different course from ordinary peritonitis because of the less extent of the pleura and because the infective organism is usually of less virulence. When equally virulent organisms are introduced into the pleural cavity the course is quite as rapidly fatal as when the peritoneum is affected.

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## CHAPTER XVII

### TREATMENT OF ACUTE GENERAL PERITONITIS

No other disease that falls to the lot of the surgeon places so heavy a burden on his judgment as does the management of acute inflammations of the peritoneum. In the progressive type it depends largely on his judgment whether or not it shall be halted in its course; or, if tending to spontaneous limitations it may be set on its way again by his injudicious manipulations. It is in this field that fate exacts from the young surgeon its heaviest toll. He may learn much from books, from experimentation and from the masters of the art, but it is under his own hands only that the minute details can be mastered. How apt a pupil he shall be depends on the inherent acuteness of his perception and the profundity of his knowledge of the fundamental principles which underlie the genesis and propagation of the inflammatory lesions of the peritoneum. If he proceeds without the former he has not heard aright the call to duty, if without the latter he commits a crime against his patient.

**Historical.**—The history of the literature bearing on the treatment of peritonitis is a long one made up, as is the history of the treatment of most diseases, of a vast deal of irrelevant papers, many case reports of value in the final summation and a few path-forming papers. It is the last group only which will receive attention here.

Accounts of incision of abscesses arising within the abdomen are recorded in the earliest medical writings. It was only when perforations through the skin threatened, however, that this procedure was undertaken. Only much later did surgeons open the abdomen to drain purulent accumulations. Chomel was one of the first to formulate rules for incision in walled-off intraperitoneal abscesses. In this early period puncture was resorted to in order to remove the pus without exposing the abdominal contents to the air. As late as 1876 Kaiser reported sixteen cases collected from the literature cured by puncture, paracentesis or incision. He advised such treat-

ment only after the acute symptoms had subsided. Schmidt advised long incision when the abscess was not well walled off in order to avoid accessory accumulations.

Relaparotomy after infection during abdominal section was advised by Tait. These papers were the first clearly to foresee the modern operative treatment for peritonitis. Leyden first suggested energetic irrigation in addition to drainage.

From this date the operative treatment of peritonitis forms a large part of surgical literature. Two important papers appeared at this time; one by Mikulicz in which he practiced suture, irrigation and immediate closure, albeit without success, and one by Krönlein in which immediate suture was followed by recovery. In 1886 Truc presented a historic review of the treatment of peritonitis to date. In this year our countryman Hall reported a case successfully treated. Bull and Gaston are names to be remembered in connection with the early treatment of this affection.

For early operation the first clear recommendation was by Bull. Simultaneous with this Sands, McMurtry, and McBurney made similar recommendations. Matter of fact as these suggestions now seem to us, they were a veritable calling in the night. To appreciate this, these papers must be read in their entirety. Following this Senn raised his voice in favor of early operation. McBurney advanced the efficiency of treatment by clarifying the diagnostic side, and first noted the point at which pain is most frequently found in impending appendiceal peritonitis.

Reichel advancing far ahead of his time discussed on experimental grounds the question of irrigation in septic peritonitis and condemned the practice in the following words: "Die spülerei der Peritonealhöhle ist eine Spielerei."

About this time Schooler released adhesive bands which produced occlusion of the gut in acute perforative peritonitis and saved his patient. Robinson in addition to incision and drainage advised the use of saline laxatives to prevent adhesions. Renvers called attention to the fact, still new to some, that operation in these patients should be carried out with expedition. In line with this Körte urged a simple procedure of incision and drainage. This is perhaps the first clear, comprehensive statement of all that is best in the treatment of peritonitis. Though the profession has

marched forward since that paper was published the advance has been in a circle.

From this time on the important literature may be discussed in connection with the presentation of the problems which confront the surgeon today and represent therefore in a way history in the making.

**Preventive Treatment.**—The chief advance in the treatment of peritonitis lies in the direction of prevention. Diseases which may be complicated by peritonitis are so managed as to lead to their cure before the disaster develops. Gastric ulcers are cured that they shall not perforate, gall stones are removed in order to obviate a suppuration of the gall bladder. Typhoid fever is so managed that the least risk of perforation shall develop. Gonorrheal tubes are allowed to cool off before being operated on in order that infection shall not be spread by the manipulation of the surgeon.

Prophylaxis finds an even more important field in preventing the extension of a localized peritonitis. In many instances the presence of a diseased state of an organ is not recognized until the peritoneum becomes inflamed. The early recognition of such inflammation and the skillful removal of the disease causing it represents the chief field of prophylaxis.

**Medical Treatment.**—The medical treatment of peritonitis has brought out an astonishing array of drugs. Surgical treatment has so overshadowed the medical treatment that few surgeons are disposed to believe that medical treatment can have any place at all. It is a mistake to be too sure about anything and it seems to me that the literature of the medical treatment is well worth reading. Sometime possibly a valuable drug will be discovered and a knowledge of past efforts will aid in recognizing it when it is found. At any rate historical knowledge always serves as a check against overestimating our own importance. Omitting those drugs of purely historical interest but two general groups remain for consideration. The first, looking to elimination, consisted of cathartics more or less drastic. Tait employed salts to prevent post-operative peritonitis and for a time a like plan was followed to prevent spreading in local forms of peritonitis. This method was pernicious in practice and has been wholly abandoned as a method of treatment. One still sees it in use in early peritonitis when the

practitioner, under the general diagnosis of gastritis or ptomaine poisoning, gives a cathartic to eliminate the toxin.

*Opium.*—The use of opium several generations ago was universal. It was supposed that it possessed a direct healing virtue. Alonzo Clark first began its use about 1840. The essential feature of the treatment is that the patient shall be completely narcotized with opium. In his first case he gave 100 grains of opium in the first four days of the treatment. He mentions a case in which he used 32 grains of morphine in twenty-four hours. In this case 208 grains of opium were given in twenty-six hours about the fifth day of the disease and on the sixth day 221 grains, on the eighth 224 grains, on the ninth and tenth days the same amounts, on the eleventh day 247 grains, and on the twelfth day 261 grains. After this with the patient improving, the amount was gradually lessened. The patient recovered. Clark calls this heroic treatment; none will offer contradiction.

This treatment was quite generally followed, though be it said mostly with less heroism. Wood, for instance, recommended that 75 grains of solid opium be given daily for five days. He notes that as the disease wears out the ability of the system to stand large doses subsides, so that the quantity must be gradually reduced.

These heroic doses have long been abandoned and the use of this drug in any dose is generally condemned. Stockton still speaks of the treatment with a measure of enthusiasm. He seconds Starling in the belief that opium tends to lessen distention of the intestines by its sedative action on the splanchnic nerves. Crile recently advised the use of morphine to lessen exhaustion.

It can not be denied that the drug is capable of fulfilling two of the offices ascribed to it by the old writers, namely, to prevent exhaustion, and to cause quiet. The dose necessary to meet these ends need be but a small fraction of those formerly advised. The question arises whether or not opium does not have some direct action in neutralizing the toxic effect of the toxins. The enormous dose formerly employed would not be tolerated in any other disease. Unfortunately no effort was made in cases where huge doses were given to recover the drug from the stools. The truth of the matter is likely that but a small amount of the drug was absorbed. In my early practice, when available surgical skill did



not seem to warrant operative treatment, I made use of the opium treatment. I discovered to my discomfiture that patients receiving large doses of opium by the mouth were proportionately vastly more susceptible to hypodermics of morphine. This difference is not due to the substitution of the alkaloid for the crude drug, for large doses of morphine may be given by the mouth without getting this effect. Opium lessens absorption from the peritoneal cavity in the normal animal and from my observation I feel certain that the ability of the patient to stand such large amounts of opium is due to the fact that the drug is not absorbed. In peritonitis absorption from the gut tract is lessened, as is evidenced by the accumulation of fluid in the intestines, and when the peritonitis subsides absorption increases and the tolerance to the drug is lessened.

One curious phenomenon was noted in a number of instances. Despite the continued exhibition of real respectable doses of opium the patient had a spontaneous movement from the bowels. There seems to be no ready explanation for this.

Modern surgery leaves but little room for the use of opium. Once the real cause has been removed, the disease progresses to recovery. After the cause is removed, there seems to be no valid objection to the use of the drug to alleviate suffering. I can not suppress the feeling that the opium treatment has been too completely abandoned.

In the extreme cases of acute diffuse peritonitis, with apprehensive look, leaky, blanched or semicyanosed skin I believe opium offers more than surgery. Opening the abdomen in such cases but augments the already deadly rate of toxic absorption. Here I am convinced opium is better than morphine hypodermically. Possibly the local action of the opium on its way down the gut tract may exert a beneficial influence. This whole question is so fraught with uncertainties that its review in competent hands is much to be desired.

*Epinephrin*.—This drug was found to stimulate the circulation by raising the blood pressure and having assumed that the pressure falls in peritonitis it was assumed it would be desirable to raise it. It was assumed also that if epinephrin would raise the pressure in normal animals it would also do so in patients with peritonitis, hence the use of epinephrin would be desirable in this disease.

Whether or not it might have some deleterious effect has not troubled clinicians. Nevertheless, it is worth recalling that Josué found that repeated injections of epinephrin in animals is followed by the formation of distinct arteriosclerotic places in the intima of the larger vessels. A considerable number of injections, as many as eighteen, are required to produce these results and Loeb and Githens did not find them to be constant. What is of much greater importance is that Erb found extensive changes in the muscle cells, consisting in the disappearance of elasticity of the elastic fibers. These observations were in a large measure confirmed by Pearce and Stanton. While inconclusive, these studies suggest the possibility that the drug may produce mischief. Certainly a drug which produces degeneration of muscle cells should be used with caution in the treatment of peritonitis. Holtzbach sought to establish a scientific basis for its use. He found that the beat of a frog's heart, poisoned by sodium arsenate, became stronger with the application of a weak solution of adrenalin.

There is no doubt that, as Peiser showed, adrenalin added to the salt solution introduced into the normal peritoneal cavity markedly lessens the rate of absorption. It does this by its vasoconstrictor action. If this drug is of any use in peritonitis the benefit from this action would seem more plausible than from its capacity to raise the blood pressure.

A number of surgeons speak enthusiastically of the value of epinephrin in the treatment of peritonitis. Meissl, Rothschild, and Heidenhain by means of experiments demonstrated that blood pressure could be heightened and so maintained by a more or less constant infusion of sodium-chloride-epinephrin solution. The last named believes that this remedy is useful in severe cases. He details one case which at first seemed inoperable and became operable after the use of this remedy. The patient subsequently recovered. Much less enthusiastic is Mummery.

Whether or not epinephrin has a place in the bridging over of a critical stage in peritonitis is difficult to say. That there is a fall in blood pressure in the terminal stages there can be no doubt. Whether this is due to a central exhaustion, a vascular dilatation, or a weakness of the heart muscle is difficult to say. Seelig's experiments indicate that it is not a central exhaustion. That it is

often, late in the course of the disease, at least in part, an expression of cardiac intoxication is likewise certain. It is possible, even probable that in an earlier stage vascular dilatation plays a prominent part. Even so it is still a question if such an ephemeral drug can exert a lasting beneficial influence. Hunter was of the opinion that a single abstraction of blood, by once lessening the load, extended its influence beyond the period of its activity. It may be so with epinephrin. Heidenhain was of the opinion that its effect lasted at least six or eight hours. On the other hand Crile and Janeway found that the pressure when raised by adrenalin fell again in a short time. My own experience in many cases, where this drug was used in conjunction with local anesthesia, indicated that the maximum pressure is maintained a very few minutes only. The conditions may be different in the peritonitic patient. Used as it is in peritonitis in conjunction with a considerable amount of salt solution it is impossible to judge to which of these the good results, if any, may be due. Its value when given alone subcutaneously certainly is negligible.

Until further light is shed on the subject for me the following situation indicates a trial; when there is distention of the intestines, with pallor or cyanosis and a large soft pulse. Then a pint or two of adrenalin-sodium-chloride solution will at least bring temporary improvement. I believe Neu is right when he insists that the solution must actually be put into a vein. He recommends the use of a glass cannula tied into a slit in the vein so that the solution may be conveniently introduced at short intervals.

*Ether.*—This drug is supposed to act as a general stimulant and as a local antiseptic. The basis for such a belief is wholly unsatisfactory. The use of this drug seems to be largely, if not entirely, empirical. Morton found that ether is first dissolved in the lymph of the tissues, then enters the blood stream and is carried to the lungs where it is eliminated, beginning as quickly as 3 or 4 minutes after it is introduced. She quotes Park as saying that colon bacilli are killed in one minute with a 75 per cent solution of ether in oil and in 10 minutes with a 50 per cent solution. Jenanneret believes it has an antiseptic action and is of peculiar virtue in that it penetrates deeply. He does not reveal the source of his information. He believes also that it acts as a tonic. He does not state

the basis of his belief. Waterhouse quotes Tapley as saying that ether is decidedly bactericidal. According to him it produces an exudate in which the endothelial cells at first predominate while later the polynuclears are the more numerous. My own studies failed to disclose any action save that the cement substance between the endothelial cells is dissolved, and in prolonged action, the cells are loosened and ecchymosis in the tissues takes place. The same action is observed to a lesser degree in the lung epithelium after prolonged inhalation anesthesia. Its action does not seem to be in any way to abstract leucocytes or to excite other evidence of reactive processes. In what way this drug might be believed to be useful in peritonitis does not appear and clinical experience seems to bear out this surmise.

Morestin's seems to be the first of a number of papers expressive of satisfaction in its use, but the details of the case reports indicate clearly that the optimistic attitude of the writers is wholly unjustified by the facts. For instance Saliba bases his belief on an experience of 248 cases. He quotes five of these in detail. In one of these the patient became suddenly pale, with thready pulse and shallow respiration, followed by basal pneumonia on the third day. Another became cyanosed fifteen minutes after leaving the operating table and remained unconscious for twelve hours. Another suffered from diarrhea for a few days. Despite this experience he regards ether as a safe and beneficial antiseptic. The amount used varied from one to three ounces. Phelip and Tartoïs noted that stupor often follows the use of ether. Santy found that 12 c.c. in a 1900 gm. rabbit produced cyanosis, dilatation of the pupils and death. Tansini used it in his two cases to wipe out feces that had escaped into the peritoneal cavity. He also uses it to wipe the line of suture after gastroenterostomy. Waterhouse regards three ounces as the maximum. Auvray is said to have left a quart of ether in the abdomen.

Pope in experiments on animals found that a dram of ether left in the peritoneum of a rabbit profoundly shocked the animal. A deep narcosis with fall in the blood pressure followed. He believes that the normal defenses of the peritoneum are broken down by ether and warns against the use of this substance in the treat-

ment of peritonitis, a conclusion which will be enthusiastically endorsed by those who have experimented with this drug.

*Camphorated Oil.*—This substance has been used for the double purpose of limiting infection by local use and as a stimulant. As a stimulant to the heart this drug no doubt has a place. There is no evidence that it is of particular value in peritonitis. Its local use in the peritoneal cavity has but slight evidence to support it. Glimm found that absorption of a sugar solution was slowed if oil was injected into the peritoneal cavity before the sugar solution was introduced. The slowing was supposed to be due to the plugging up of the lymphatics by the oil globules, it being assumed that a slowing of absorption was desirable. Pfannenstiel was the first to propose camphorated oil as a prophylactic measure. He used 50 to 300 c.c. of olive oil in 20 cases and 25 to 50 c.c. of a 10 per cent camphor in olive oil in 22 cases, and a preoperative intraperitoneal injection of olive oil in 120 cases. He used 30 to 50 c.c. of 1 to 10 per cent of camphorated oil injected into the peritoneal cavity four days before the operation. The reaction so produced lasted from two to seven weeks. Burekhardt used 50 to 100 g. as a prophylactic measure. Much greater proportions of camphor have been used. Hœhne used 10 per cent. Even this did not prevent collapse from the oil, for Rübsamen had a fatal collapse after the use of 170 g. of 10 per cent camphorated oil. Schepelmann found that in experimental peritonitis the animals died more quickly when camphorated oil was used.

The danger of using oil in the peritoneal cavity has already been discussed in the chapter on the prevention of adhesions. The addition of camphor seems not to lessen the danger to more than a very slight extent. Animal experimentation and clinical experience are in accord in teaching that oil in any form should be avoided in abdominal surgery.

**External Application.**—*Heat.*—Local application of heat, particularly moist heat, has long been used for the relief of abdominal pain. In the early stages of peritonitis it aids materially in relieving such pain as may be due to the spasmodic contractions of hollow organs. It is the most harmless palliative means and can be advantageously employed during the period the patient is observed for the purpose of making a diagnosis.

**Dry Heat.**—The ubiquitous hot water bottle furnishes the most convenient but the least effective means of applying heat. As a direct means of controlling peritonitis its use is of quite recent date. Gelinski was the first to employ it systematically as a measure of after-treatment in peritonitis. He employed dry heat in the well-known Bier's oven. Danielsen warned against its general application. Strumpel advised a temperature up to 550° C. Iselin believes that drainage is promoted by the hot air bath.

**Moist Heat.**—Moist heat is much more efficacious in relieving pain from inflammation or distention than dry heat. Pads as large as the abdomen are made up of half a dozen or more layers of flannel and after being wrung out of hot water are applied to the abdomen. These may be covered with dry blankets to retain the heat. Moist heat is enhanced in effectiveness if its stimulating effect is augmented by irritating chemicals. Turpentine and cantharides are usually employed. Turpentine is most used, a dram being sprinkled over a hot pack prepared as above noted. Hot packs act on the general principle of counter-irritants and are often very effective against distention of the abdomen.

**Ice Pack.**—In the beginning of an acute peritonitis the ice pack often lessens the pain and is supposed to lessen the reactive processes. If the patient is required to balance an ice pack on his abdomen he is at least restricting his movements.

### Operative Treatment

Once the diagnosis of peritonitis is made the question of its disposition to spread must be considered. Most cases tend to spread, therefore the advisability of operative intervention must be considered.

**Indications for Operation.**—This entire monograph is an attempt to answer this question for one individual surgeon. It is a situation in which the surgeon must take stock of himself as well as of the patient. My teacher of obstetrics, the late W. W. Jaggard, was wont to say that before any operation is done the surgeon should ask himself, "What harm may I do?" There is no department of surgery in which this question is fraught with so much responsibility as in acute peritonitis.

For the so-called occasional operator my advice is, don't do it.

Many cases of stormy beginning subside and localize. Then he may operate. Those cases which have no tendency to localize, such as perforating ulcers of any sort are seldom improved by unskilled hands. In traumatic perforations, before inflammation begins, the amateur may be of service. It is in inflammations which tend to localize, such as most cases of appendicitis and all the gonorrheal perisalpingitides, that injudicious operations do most harm. Operations lasting two or more hours are still committed in acute appendicitis. There can be no question but that the patient is menaced by such prolonged manipulations. It was my privilege to have practiced when it was the rule to await the interval for operations for appendicitis. I have also lived in an environment when everybody operated on appendicitis patients "as soon as the diagnosis is made" and alas, often before. As a result of these operations I have concluded that the chief indication for operation in acute peritonitis is the arrival of a surgeon. The requisite skill being available, an acute spreading peritonitis is an indication for operation in many instances. The offending lesion may permit of removal and the mischief done then is capable of neutralization in the shortest possible time. The old adage, too late for early operation and too early for late operation, has lost its significance in the development of our knowledge of the management of these cases. There is no rule that can be written that can guide a trained surgeon.

There comes a time in the spreading type when operative procedures offer little or nothing. Pinched features, cold, clammy skin, blue extremities, thready pulse, spell impending disaster and incision can but hasten the end. Whether some such cases are capable of spontaneous localization of the process with subsequent recovery is a matter of study. That absorption in a distended abdomen is hastened by incision there can be no doubt.

**Preparatory.**—The patient afflicted with acute peritonitis should be prepared for his operation in the simplest manner possible. The elaborate preparations permissible preliminary to operations on noninfected patients are out of place here. Food and drink should be withheld. If there is vomiting this may be controlled with morphine while the preliminary steps of preparation are being completed. Some surgeons practice gastric lavage. In all conditions

except perforative lesions of the stomach this is permissible and if it can be accomplished without retching on the part of the patient it is actually indicated, but too often the strength required to accomplish it does harm in spreading the infection. In such instances it had best be deferred until the conclusion of the operation.

**Preparation of the Skin.**—Manipulation of the field of operation is distressing to the patient and may do harm if rigorously employed when the patient is asleep. Dry shaving, when needed, followed by the application of tincture of iodine is the work of but a moment and meets every requirement in these cases. In the less acute cases cleansing with soap and water before as well as after shaving is the ideal method. The use of a sterile dressing after the preliminary cleansing is not objectionable except as it tends to continuously remind the patient of the coming operation. It is quite useless, however, for no more infections occur without it than with it. The use of a moist dressing, once in vogue, has now been universally abandoned. Painting the skin with iodine before the operation meets every requirement.

**Anesthetic.**—An acute peritonitis hypothecates a generalized symptomatology but with local lesion. Because of the general state of the patient the operation should be done in the least time possible. These requirements demand a general anesthetic since the extent of the manipulation can not be foretold before the lesion is exposed. Local anesthesia followed by gas may be employed when a general anesthetic is contraindicated. Usually however these patients are nervous and irritable and any attempt at infiltration anesthesia may disturb them. Gas alone where the lesion is simple may suffice, or if a more complicated lesion is encountered, ether may follow. Ether is usually the ideal anesthetic, but its use should be restricted to the smallest amount compatible with the unhindered manipulations of the surgeon. Timid anesthetists often seriously hinder the work of the surgeon by a failure to push the point of relaxation. By so doing they prolong the time of the operation so that in the aggregate more anesthetic is consumed than if a bolder exhibition were practiced. Chloroform, because of the danger at the time of the operation and particularly because of the possibility of late yellow atrophy of the liver, which is apt to follow its use in infected patients, is contraindicated.



**Time of Operation.**—In some cases immediate operation is demanded irrespective of the surroundings, and in a large measure irrespective of the qualifications of the operator. This includes those cases of acute peritonitis due to perforations of the gut where there has been no anticipatory reaction on the part of the peritoneum such as perforative gastric or duodenal and typhoid ulcers, those incident to intestinal occlusion, etc. The rupture of encapsulated abscesses into the free peritoneal cavity may be included in this list.

Those cases in which there is a gradual involvement of the peritoneum from disease arising in the wall of a viscus and gradually approaching the surface do not always demand immediate operation. In this group appendicitis is preeminent, followed by cholecystitis and salpingitis. In these cases there is a range of opinion in selecting the time of operation. The average patient afflicted with one of these diseases runs less risk from his disease than from the operation by an inexperienced operator. Very unfavorable surroundings may influence the experienced operator to elect to operate at some other time.

**Site of the Incision.**—Two factors must be considered in selecting the site for incision in the operation for acute peritonitis. The first is accessibility to the site of the lesion and the other the prevention of infection of the general peritoneal cavity. In acute lesions in which walling off is not to be expected accessibility, generally speaking, is the dominating factor, while later when there is partial or complete walling off the second factor is the more important. For instance in perforation of a duodenal ulcer or in the beginning of an acute appendiceal peritonitis the route which makes the site of the lesion most easily accessible may be selected, keeping in mind of course the fundamental principles of operative surgery; while in appendicitis partially or completely walled off the incision would be made lateral to the focus of infection in order to avoid disseminating the infection into the unaffected portion of the peritoneal cavity. The more complete the walling off and the more virulent the infection the more important does this rule become. A localized pelvic peritonitis may demand drainage through the rectum or vagina, and one situated below the diaphragm may demand a transpleural drainage.

From the foregoing it is evident that the character of the infection, its stage of development, and its topographic relations must be taken into account in selecting the site of the incision. Obviously therefore this question can be adequately discussed only in consideration of specific groups of cases; indeed in detail only when a concrete case is at hand in which all the questions in pathogenesis can be determined.

**Management of the Exudate.**—When an infection has produced a purulent exudate in any region of the peritoneal cavity the fundamental problem is to effect its removal. If an accumulation is walled off the contents are under equal pressure from all sides and if an opening is made at any point the fluid escapes because of the elasticity of the walls. When there is no walling off no such pressure exists and the force required to propel the fluid must be manually supplied. This may be done by sponging or by irrigation. Even in the diffuse variety the general intraabdominal tension tends to force fluid out of an opening, whether dependent or not.

**Sponging.**—Because of the capillarity of gauze pledgets fluid is taken up by them and when saturated they may be discarded. This act is repeated until all of the fluid is removed. The difficulty met in employing this method is dependent entirely on the amount of the exudate and the extent of the area involved. When gently done this method has the advantage of not disseminating the infection.

**Irrigation of the Peritoneal Cavity.**—When one sees a deleterious substance it is quite natural that one should desire to wash it off. This natural desire has found expression in the treatment of peritonitis. The central thought is to remove the toxic material by means of irrigation and thus prevent its absorption into the circulation. The logic is good, the result disastrous as is so often the case when therapeutic problems are decided by abstract considerations. The fallacy lies in the fact that the toxins being absorbed are not so much those contained in the fluid free in the abdominal cavity as those in the tissues themselves, which are not reached by the irrigating stream. The chief mischief was done by distributing toxic material over areas of peritoneum not previously involved.

The general plan was to introduce large amounts of normal saline solution into the peritoneal cavity which when it returned was supposed to carry the infected material with it. Since the action was largely mechanical it was but natural to suppose that the larger the amount used the more efficacious the treatment.

The fundamental fault of irrigation lies in the fact that the indifferent fluid tends to carry the infective material to regions not previously affected. Much harm also is done by removing the natural defensive forces of the tissues. The exudate covering the surface of the peritoneum is composed of serum and cells and in just the measure that irrigation is effective it is harmful by removing this protective measure. The maximum of mischief was done by adding manual friction to the irrigation.

In localized abscesses irrigation is sometimes employed for esthetic reasons. Offensive discharges may be deodorized by irrigation with potassium permanganate. Irritative discharges may be removed by irrigation in some instances.

Some weird theories were advanced to explain the hypothetic benefit from irrigation. For instance, salines were supposed to mechanically remove the toxin-laden pus and at the same time were supposed to be absorbed, thereby stimulating the vital functions and promoting elimination.

Though generally abandoned, irrigation has been recommended from time to time by very able surgeons. Blake developed abdominal lavage to the highest degree. He employed an irrigation tube which by means of a syphon arrangement sucked the fluid out as completely as possible. Crandon and Scannell also describe a special apparatus. Mikulicz believed that the mechanical cleansing was useful and that the fluid remaining produced a hyperleucocytosis and acted as a stimulant as well.

There are still a few relatively recent papers which advocate its use. Among those which may be mentioned are Schmidt, Propping and Iselin.

Reichel was one of the first to oppose irrigation. He declared that "Spülerei ist eine Spielerei." Many have opposed it, and since Murphy excluded it from his method of treatment, it is but seldom used. Robinson's excellent work along the same line has gone unheeded.

While clinical experience was the chief factor in eliminating irrigation from the recognized means of treatment experimental evidence added salve to the process of elimination. Clairmont and Haberer in experiments on rabbits found that irrigation did not delay, on the contrary seemed actually to hasten death in experimental peritonitis. Noetzel likewise failed to find any experimental evidence that irrigation was useful. There is a question whether or not irrigation may not be useful in the face of impending paralysis of the bowel. Holtz showed that a gut that had almost ceased to move by stimuli became active again after irrigation with saline solution. This is capable of but momentary stimulation, however.

There is one condition in which irrigation seems rational. In instances where large amounts of fluid have been poured into the peritoneal cavity, as from a recently perforated ulcer or wound, it seems that irrigation may be useful by removing mechanically foreign bodies which have escaped from the intestinal lumen. There is no doubt that peritonitis is much enhanced when foreign bodies enter along with bacteria present. If the manual force of a stream of fluid can be made to remove them before inflammation has been set up, the irrigation may be beneficial.

**Drainage.**—The term drainage in a general sense signifies the institution of such relations that stagnant fluids shall be made to escape by the force of gravity. As applied to the abdominal cavity this meaning of the word must receive distinct modification. Here the hydrostatics are much modified by the intraabdominal pressure and the agglutinations and adhesions of the abdominal organs to each other which commonly take place in conditions demanding drainage. In general hydrostatics drainage demands that the cavity containing fluid shall be opened at its lowest point. This requirement can rarely be met in abdominal drainage. We must, therefore, utilize the intraabdominal pressure, and other forces that will overcome the attraction of gravitation. The problems in drainage have been nowhere so well stated as by Yates. He states, "If drainage of the peritoneal cavity is possible it is limited by—1" the time requisite to the functional seclusion of the drain through (a) the close application of serous surfaces to the drain and (b) its subsequent encapsulation in adhesions; and 2nd by the physical laws governing (a) the removal of the drainage

material from the tube, and (b) the restitution (absorption) of the capillary action of the gauze." These may be discussed under the respective headings.

It is interesting to note that it was J. Marion Sims who first proposed drainage after laparotomy. He advised particularly the vaginal drain. It is interesting also to note that he referred to the drain as a plug the purpose of which was to hold the edges of the wound apart. He therefore started at a point which we have not yet reached.

*Gravity.*—The rule that fluid collections shall be opened at their lowest point can but rarely be realized neither is it of importance. It is only when the fluid is contained in a cavity with fixed walls that this becomes of moment. When there is a collection of fluid in the pelvis an opening at its lowest point facilitates the escape of its contents. For the abdominal cavity drainage at the lowest point is but seldom striven for.

Obviously there are anatomic difficulties in the way of draining the abdominal cavity at its lowest point. Attempts have been made to compensate for this by so placing the patient that the wound area would become the most dependent part of the body. In order to reach this end the patient has been set up, turned on his side and even completely over, belly down.

The abdominal cavity is not a vat, but a cavity containing organs. The laws of physiology and not the laws of hydrostatics are those that must be studied in attempting to solve the problem of drainage.

*Viscosity of the Fluid.*—One of the fundamental reasons that the fluid within the peritoneal cavity does not respond to the force of gravity is because the cohesion of its several particles exerts the greater force. When in great mass of course the bulk of the fluid may escape, but it leaves a thin layer lying next to the peritoneum. Since the intoxication takes place in the region of the peritoneum, the escape of the bulk of the fluid makes little difference. In some instances the exudate is formed by a diphtheroid membrane the removal of which is difficult by manual means and is wholly uninfluenced by gravity.

*Duration of the Drainage.*—The idea of drainage assumes that the exudate to be conducted away is in a fluid state and that there

shall be no mechanical hindrance to its outflow. At the time of the operation those conditions are met in diffuse peritonitides of sudden onset, such as perforating ulcers, and occasionally in appendicitis. Once the fluid comes in contact with the air and the viscera come in contact with the drainage tubes all this quickly changes. The course the drainage process then pursues varies with each case.

Experimental evidence on the duration of time a drain remains effective is of general interest only, for the conditions obtaining clinically can not be reproduced in the experimental animal. In experiments the duration varies much with the material used for drainage and the fluid experimented with. Gauze excites adhesions in the abdomen of a normal animal, beginning in the course of an hour or two, they are quite well walled in within eight hours, so that the drainage ceases. Murphy places the extreme limit of efficiency at 18 hours. This author conceived the clever idea of placing a colored gelatin solution within the peritoneal cavity. This substance, fluid at the body temperature, becomes solid when cooled permitting the study at leisure of the portion remaining in the abdomen. I employed an albumin solution, both with and without methylene blue, of the general specific gravity of thin pus. This substance drains less than eight hours. Animals whose blood has been made incoagulable drain a longer time. In recent studies Petroff obtained results corresponding very closely to my own. He placed tampons in the abdominal cavity of rabbits. After an hour or two a solution of methylene blue was injected into the peritoneal cavity. Drainage diminished after 5 or 6 hours. In all of these experiments the fluid escaped from the wound around the tampon and was not conducted out by the gauze. In other words the gauze acted only as a plug to hold the edges of the wound apart. Ward and Robb found drainage to be effective for twenty-four hours and McGuire placed the time at forty-eight hours. Sanger placed the time at a day longer. V. Gubaroff found that the drain became enclosed in twenty-four hours and in less time if inflammation was present. Delbet found that the drain was completely enclosed in a neomembrane in forty-eight hours. Rubber protective remains effective for a much longer period, usually a day or two. It is interesting to note that the cigarette drain with the protruding tuft of gauze becomes walled in as quickly as does the gauze

drain. Murphy places the limit at three days. Glass tubes remain patent for a longer period, being occluded by a fibrin plug sooner or later. The manner of walling off depends on the situation. When within the range of the great omentum, it is this organ that walls in the foreign body. In other situations the neighboring organs accomplish this act. When in contact with solid parenchymatous organs there is much delay. Between the liver and diaphragm, for instance, the drainage is effective several times as long as in the region where the great omentum can act.

Under clinical conditions the duration of drainage varies under wide limits. The more fluid the exudate, the longer the drainage will continue, and the less capable the tissue is of reacting the longer the drainage. In virulent streptococcic and pyocyanus infections where all efforts at adhesion formation are nullified drainage may continue for a long period. In very toxic states of the patient drainage may remain unhindered for several days. On the other hand in conditions in which the walling-off processes are already far advanced the duration may be very short for adhesions form in a few hours. Fluids rich in fibrin quickly surround the drain and impede further outflow. When both these factors are active drainage may cease in half an hour. In walled-off abscess foci also the duration is dependent on the amount and character of the exudate. On the whole the period of drainage is less than in diffuse inflammations because the factors going to limit it are active, for it was by virtue of these that the abscess became walled off.

*Mechanism of the Drain.*—A gauze drain acts by its capillarity. This function ceases as soon as the meshes of the gauze become filled with coagulated lymph. This period is measured by hours. After this time the drain acts as a plug serving only to keep the edges of the wound apart, allowing drainage to take place around the gauze. The gauze tends further to limit its usefulness by irritating the surrounding tissues to form adhesions. At the same time by irritating the edge of the wound the gauze excites an exudate about the tube which tends to keep the wound open.

The tubular drain allows the fluid to flow through its lumen. The efficiency is dependent on the size and composition of the tube and the character of the fluid to be conducted away. The forces operative in causing fluid to flow from the tube are gravity and

the expansion of the intraabdominal tissue, reducing the space available for the fluid. Mikulicz was the first to point out the dependence of tube drainage on intraabdominal pressure. Because fluid is escaping from the tube is no evidence, however, that fluid from the general peritoneal cavity is being conducted away. The tube by its presence may excite an exudate which may then escape through the tube. This is merely a reaction of the tissues about the wound against the foreign body.

*Factors Which May Aid Flow.*—Theoretically anything that would prevent the coagulation of the exudate should increase the flow. Wright's solution has been proposed by Crandon for this purpose. The calcium in this solution is supposed to prevent coagulation while the hypertonic salt solution (4 per cent) is supposed to increase osmosis. Whether or not this works out in practice is difficult to say. Wet dressings of any sort may prevent the pasting in of the tube in the wound and may perform some service. Hot moist dressings applied over a recently drained wound probably are the most efficient means of promoting drainage.

*Dangers of the Drain.*—By causing adhesions between coils of gut the drain may foster the development of an intestinal obstruction. Gauze is more apt than other drains to produce this result. Because of this danger the drain should be placed about the periphery of the abdominal cavity whenever possible so that a loop of gut shall not become adherent to it. The presence of a drain tends to aid the formation of permanent adhesions and thus to leave a permanent disability. Placed near a line of suture or ligation by exciting a flow of serum away from the stitches the healing is interfered with and the establishment of a fistula is made more liable.

By pressure on a vessel, either in the abdominal wall or in the walls of the organ, erosion of its walls may result and a secondary hemorrhage ensue. Turner advises that the drainage tube should not be left in contact with a tissue bearing on an important vessel more than two or three days. Aside from hemorrhage, pressure of the tube may cause inflammation of the vessel wall with thrombosis. This may become the starting point for any one of a number of disasters.

Pressure of the drain against a hollow viscus may result in per-



foration. A firm substance is more apt to act this way than a pliable one. It is this danger that mitigates strongly against the glass drain. The erosion of vessels is most apt to occur about the tenth day. When these firm-walled drains are removed in the course of a day or two this danger does not obtain.

The loss of the drainage tube is one of its most serious dangers. It may slip entirely within the wound and escape. It is only by the most rigid care that these accidents can be reduced to a minimum.

*The Removal of the Drain.*—The time for the removal of a drain is dependent on the material used, the purpose for which it is employed and the qualitative character of the infection. The first two factors admit of a measure of abstract consideration but the last must be judged in the concrete case.

The time of removal is dependent also somewhat on the material used.

A gauze drain after a day or two becomes firmly attached to the edges of the wound by the formation of fibrin about the gauze. Fine processes extend into its meshes firmly fixing it to the wound. During the succeeding days a forcible removal produces a considerable injury. By the eighth day the granulations have developed sufficiently to destroy these fibrin bundles and the gauze is again loosened and the removal becomes easy. Those portions of the wound most capable of producing granulations loosen the drain earliest. It is advantageous sometimes to remove that portion only which has become loosened. Rubber drains offer no attachment to fibrin bundles and consequently are easily removed at any time. When allowed to remain for some time granulation tissue may grow into fenestra and thus fix the tube. When the openings are large a segment of gut wall may extend into them and the forcible removal of the tube may produce serious mischief.

*Material Used.*—From the foregoing it is apparent that the agent employed for drainage is dependent upon the fundamental purpose in view. When a purely mechanical factor to remove exudate is desired, a smooth substance little calculated to excite adhesions is indicated. Rubber tubing is ordinarily employed, though glass was formerly extensively used and is theoretically the better because it irritates the tissues less. When adhesions are to be invited in order to wall in the infected area and the drainage is

but incidental, an irritating substance such as gauze should be employed.

*Glass Drain.*—This material was first introduced by Koeberle. The advantage of glass lies in the fact that it is readily sterilized, readily introduced and does not collapse and shut off the lumen. The great disadvantage lies in its inflexibility. Because of this it may be uncomfortable to the patient and may endanger some structure upon which it presses and it may become broken. For these reasons glass is now rarely employed in abdominal surgery.

*Rubber Drain.*—Chassaignac first introduced rubber tubing to avoid the disadvantages enumerated for the glass tubes. Rubber drainage tubes or some modifications of them are now nearly universally used when a tubular drain is required. They have the advantage of retaining their lumen, if one suited to the case at hand be selected, and yet present a degree of flexibility sufficient to prevent injury to surrounding tissue by pressure. In using a rubber drain a size commensurate with the amount and character of the fluid to be removed must be selected. In deep exudates as in the pelvis or under the liver, tubes with a lumen of a centimeter or more should be selected. When the exudate is widely distributed several must be employed perhaps introduced through multiple incisions. When the focus is small, the infection but little virulent, as about the stump of a subacute appendix, a small drain is sufficient. It should be so placed that a vessel is not pressed upon and the tip must not come to lie too near the site of suture if there be one in the wall of the gut. Above all it must be securely fastened in place in order that it may not escape. Its lumen may be kept patent by producing suction with a syringe at intervals. So long as this act is not done with violence it may be regarded as a harmless procedure. Usually the tube is quickly walled in and suction produces little that is deleterious to the patient. Small fenestrae are usually cut into the sides of the tube so that fluid may enter its lumen at several points, and that its efficiency shall not be wholly destroyed should the terminal opening become occluded.

The ideal use for the rubber drain is where the amount of fluid to be removed is large and adhesions are absent or but imperfectly formed, and the nature of the infection is such that effectual walling off is not to be expected. The ideal use of the tube is seen in

perforating duodenal ulcers, and to a less extent in infections from the appendix when it is spreading across the pelvis to the left iliac fossa. The tubular drain is useful when there are well walled-off abscesses as about the appendix. The tube remains a ready exit for the secretion of the pyogenic membrane while the walls are collapsing.

The rubber drain in some of its forms is likewise advantageous when drainage is desired a few hours only. The advantage lies in its smooth nonpenetrable surface which does not permit it to become attached to the wound. This finds most frequent use where a noninfected exudate may become troublesome, as where there is much oozing after separation of adhesions, or where there is a mild infection and the operator is not sure whether or not the tissue will be able to cope with it, as often occurs in very early or subacute appendicitis.

Many modifications of the rubber drain have been suggested. Applying it in a direct line or spirally has often been resorted to. Half or a fourth of the circumference of the tube has been employed where temporary drainage is desired. Peple modifies the rubber drains by placing a plicated rubber dam within a split rubber tube.

*The Cigarette Drain.*—An attempt to retain the advantages of the rubber tube and secure the advantage of a gauze drain led Kehrer to combine the two. It was sought to accomplish this by enclosing a wick of gauze in sheet rubber, making the so-called cigarette drain. For reasons unexplained this combination has become the most popular form of drain. This is surprising because it possesses the disadvantages of both and the advantages of neither. The idea of its construction was that the capillarity of the gauze would conduct fluid when there was insufficient pressure to force the fluid out of the tube and gravity could not be brought into play. As a matter of fact, as usually made this capillary action is defeated by tying a string about each end to hold the rubber about the gauze. Properly made, a string of gauze is wrapped in a sheet of rubber protective without the aid of a constricting string. If it is desired to fasten the rubber it should be done by means of sutures taking in only the rubber tissue, allowing the gauze to remain free from pressure. The character of fluid to be drained is

usually such that when it gains the meshes of the gauze it rapidly coagulates, producing a fibrinous plug thus occluding the drain. As ordinarily employed the so-called cigarette drain is equal in efficiency to a sterilized corn cob, serving the sole purpose of preventing an agglutination of the wound edges. It serves only as a plug whose sole advantage lies in its ease of removal. When a tuft of gauze is allowed to protrude from beyond the rubber covering even this advantage is forfeited.

*The Gauze Drain.*—A strip of gauze in an abdominal wound has for its primary effect the removal of fluids by virtue of its capillarity. The length of time this action takes is dependent upon the character of fluid involved. The time limit may be placed anywhere between one-half and twelve hours, with an average nearer the former than the latter limit as has already been discussed. Some surgeons seek to prolong the action of the drain by changing it at intervals. Curtis for instance recommends this procedure and he invented an instrument to facilitate the reintroduction of the gauze. I regret to note that I also invented an instrument for this same purpose, albeit in my early youth. The change of gauze is a useless annoyance to the patient since it but serves to keep open the canal that has been formed about it, and once removed there can be no excuse for its reintroduction.

Its second and most important action is secured by virtue of its irritating properties to the peritoneal surface with which it comes in contact. An exudate is excited by virtue of which adhesions about the gauze are produced. This tends to wall off the area about the tube. For this reason a gauze drain should be placed near the border line between the infected area and the noninfected area. Adhesions form in response to the irritation from the gauze and by this walling-in process the infection is isolated. Once this is accomplished there is danger of removing the drain too early. The gauze may at this time form a part of the protecting wall and, if removed, may permit the spreading of the infection through the defect left after its removal. Even if the gauze does not participate in the formation of the wall, its forcible removal may lacerate the protecting wall and thus permit an escape of infection. Therefore when gauze is used for the deliberate purpose of hastening the formation of a retaining wall, it should be allowed to

remain until it has begun to separate spontaneously in the manner already mentioned. This occurs only after the fibrin bands are absorbed, usually requiring from six to ten days, when the gauze can be removed without violence. It is true that after the gauze drain has become saturated with fibrin-forming material it becomes merely a plug and not a drain but it still performs a function by keeping the edges of the incision in the abdomen from closing too soon. During this time the deeper portions of the gauze may be performing valiant service in the forming of a protecting wall.

The ideal place, therefore, for a gauze drain is where there is an infection with a moderate exudate and a poorly limited peritonitis. It is sufficient in such cases to carry off the obnoxious fluid and to aid in forming the barrier of adhesions. It is particularly useful about a pericholecystitis or about a recently perforated appendix.

Where there is no limitation and abundant exudation gauze and rubber drains may be combined, for instance, in an extending peritonitis from a ruptured appendix with much exudate. Here a large tube in the pelvis to carry off the free fluid and a gauze drain at the border of the infection makes the ideal combination. The rubber drain is removed when the extensive exudate ceases, while the gauze is allowed to remain until the walling off is complete.

*Tampon Drainage.*—The protecting effect of the gauze drain finds its maximum development in this method. The primary object here is to stimulate the complete walling-off process so desirable in infections. Mikulicz first treated this problem comprehensively. Lennander more recently has dilated on the advantages of this method. The principle involved is that when an infected focus is not walled off from the general peritoneal cavity the barrier is completed by means of some artificial substance. Since an irritating substance is required gauze is usually selected. Organs coming in contact with this foreign body become adherent to the gauze and to each other. Mikulicz placed a large piece of gauze in an infected area which was made to serve as a sort of sac. This sac was then filled with strips of gauze until the desired bulk was obtained. The advantages of this method lay in that the gauze strips within the sac

could be removed without disturbing the surrounding tissues. The removal of the sac was facilitated by attaching a string before introducing it to what was to become its lowest point. By making traction on this string the lowest point was removed first. This method is not used now, but the general principles underlying it should not be forgotten. The same principles have been used in appendiceal infection lying deep in the abdomen imperfectly surrounded by adhesions. Van Hook has developed this plan with ideal but unnecessary completeness. An infected gall bladder may often be surrounded in the same way with advantage.

The same procedure may be applied in rare instances when there is bloody oozing with or without infection, as for instance when pus tubes are forcibly torn from the floor of the pelvis, as Coffey has recently suggested, Lennander advises the use of this method when large areas of serosa are so damaged that continued supuration is likely and when there is necrotic tissue which must separate before healing can be completed. Mikulicz advises tampon drainage about suture lines so that should the line give way they are very certain to have a barrier about the gauze which will conduct the infection to the exterior. The disadvantage of this procedure, as stated elsewhere, is that the healing of the suture line is much jeopardized by the presence of gauze and when so used fistula is almost certain to follow. If drainage is used it must be placed away from the line of suture.

Once a tampon drainage is applied it should be allowed to remain until the fibrin begins to loosen, that is, the same laws governing the removal of gauze drains must be followed.

The use of this tampon drain should be restricted as much as possible, for since it keeps a large part of the wound open for a long period, thus prolonging convalescence, it is very apt to result in a scar hernia.

*The After-treatment of a Drainage Wound.*—After the drain is removed the edges of the wound may be in part closed by suture if the drainage tube is removed within a day or two. After that time the approximation of the edges of the wound by means of adhesive strips is advisable. While this method does not equal the suture in efficiency there is less risk of enclosing an undesirable

exudate. Another great advantage of the adhesive strips is that they can be applied without discomfort to the patient.

*Fate of the Scar Following a Drainage Wound.*—In many instances the abdominal wound may close by first intention throughout its extent except where the edges are actually held apart by the drain. That part of the wound occupied by the drain closes by secondary intention. If the area kept open by the drain is small, usually sufficient firmness of scar will be produced to prevent a hernia. If the wound is large, the scar tends to become stretched after a time and a hernia is produced. One factor in the production of hernia therefore is the extent of wound left open by the drain. The other important factor is the length of time the wound is kept open. A hernia is more apt to occur after a streptococcic than after a staphylococcic infection. Old persons are more apt to develop a hernia than children and young adults.

When a wound stretches, it usually does so in from a month to several years, usually in from three to six months. Once it begins to stretch it usually continues to do so. Waiting for the scar to contract is folly.

*Posture of the Patient.*—In harmony with the dictum that all pus cavities should be drained at their lowest point, surgeons have sought to make the available opening the lowest point by varying the position of the patient. Obviously the logical thing to do was to place the patient up-side-down. Kehrer, in harmony with this, proposed that the patient be placed in the ventral position. Unfortunately patients after lying on a fresh abdominal wound for a time, lose their respect for logic, and begin to express a desire for a more comfortable position. Nevertheless from time to time other surgeons have revived for a time this position. The latest of these is Hill. As a modification of this position Coffey recommended the lateral position when the infection occupied the flanks. Dandy and Rountree likewise recommended this position.

Fowler advised the elevation of the head and trunk in order to facilitate drainage. Bode suggested the elevated head position about the same time. It is worthy to note that Fowler advised an elevation of 12 to 15 inches. The "sitting position" was the product of other minds, probably on the principle that if a little was good, a lot more should be better. Fowler was led to place his pa-

tients in this position because he had observed that patients placed in bed with elevated head to lessen postoperative nausea were marked by especial freedom from complications. It was left for other surgeons to unearth the fallacy that absorption took place chiefly through the diaphragm, as a reason for placing patients in this position. I wrote in 1909 as follows: "That there are special openings in the diaphragm making absorption here more rapid, and that there is a stream of fluid flowing toward the diaphragm, is one of the curious fallacies perpetuated in modern surgical literature. The stomata have finally been accorded a much deserved oblivion, and the notions quoted above will share an equal fate as soon as surgeons shall take the trouble to make some very simple studies in the anatomy of the peritoneum. When this occurs the Fowler position will have lost one of its most important functions." Time evidently has produced the result that I predicted would be derived from a more extended knowledge of the peritoneum.

The argument underlying all postural methods is that fluids flow down hill. This is opposed by the counterlaw that they do not do so when opposed by forces which resist this tendency. The abdominal cavity is divided off normally into a number of cavities. The intestinal coils act as dams to the movements of fluids and it is only when the amount of fluid becomes great enough to flow over these obstructions that the fluid is able to flow out by the aid of the force of gravity. When this force has ceased to act there is still much fluid retained in the abdominal cavity. This is true even of drainage in the ventral position. Intraabdominal tension is at all times a more powerful factor than gravity in expelling fluid from the peritoneal cavity. All these points may easily be tested out by animal experimentation. To attempt to do so by experiments on the abdominal cavity of cadavers must lead to erroneous conclusions for the rigid gut and abdominal walls in no wise resemble the conditions in the living patient.

In the presence of inflammation the problem is much complicated. In addition to the complication presented by the hollow organs drainage is interfered with by the presence of adhesions. In this regard each case is a problem in itself. On the whole posture can aid but little. What little it can aid is confined to the first



few hours. After that time the drainage opening becomes walled off and nothing can escape from a distance. It is useless, therefore, to subject the patient for days to an uncomfortable position. Immediately after the incision is made, posture may aid the escape of an excess of fluid. There still remains much in contact with the peritoneal surface where the actual conflict is going on. The patient may have his shoulders raised or he may be placed on his side if this is more comfortable, but to keep the patient in an uncomfortable position for days is without excuse.

**Management of Complications.**—After an operation for peritonitis phenomena develop which are unpleasant for the patient, sometimes dangerous.

**After-pain.**—By after-pain we understand any discomfort the patient may suffer after an operation. The wound in the abdomen gives rise to pain lasting 3 to 6 hours. When drains are left in the abdomen they may add to the pain. After-pain may be controlled by the use of morphine—a dose or two on the day of the operation in simple cases, while in the more diffuse or spreading varieties the repeated use of an opiate is needed. It is in the use of opium here that much difference of opinion exists. I believe it may be used to the point of securing comfort once the cause of peritonitis is controlled. Food should be withheld as long as opiates are being given.

**Gas.**—Pain from accumulation of gas within the gut tract furnishes one of the most constant phenomena of the operation. Turpentine stupes are often of use, the judicious use of laxatives may do good, their injudicious use no doubt often does harm. The control of the formation of gas by a control of the diet is better than to try to lessen the gas with laxatives. A rule to have the bowels move on the second day is often a cause for gas. If the patient is doing well, the day on which the bowels are to move is quite immaterial. Enemas may succeed in removing some of the gas. Soap suds or turpentine enemas are most apt to be effective in gaseous distention. Hypophyseal extract is used in gaseous distention. It sometimes works well, but it should not be used as a routine measure; for if the object to be overcome is too great for the stimulated contraction of the gut, harm must result.

**Management of Ileus.**—One of the fundamental factors adopted

by nature in the localization of an infective process is the immobilization of the bowels. Coincident with the immobilization is a degree of distention. This distention aids very materially in forming the barrier against the advance of infective exudates. This state is purposive and there is no better example than this of adaptive factors in pathology. It is a common error to combat this initial distention by means of cathartics and enemas. This preliminary distention usually lasts from one to seven days or even more, depending on the time required for the inflammatory lesion to localize. One of the hardest problems the young surgeon has to determine is when a pathologic state begins. When a pathologic state is hypothecated it must be determined whether the disturbance is dynamic or whether there is an intestinal obstruction present. These types must be considered separately.

*Dynamic Ileus.*—The dividing line between this purposive state and the deleterious paralysis is best determined by observing the expulsive efforts of the stomach. Postoperative vomiting may continue for a day but if it continues beyond this time the patient must be carefully observed. If vomiting ceases once and then begins again it is an omen of grave significance. Even early recurrent vomiting with extreme distention undoubtedly places it in the category of impending ileus. This vomiting is due to reversed peristalsis and may be distinguished from the reflex kind seen soon after the operation by the larger amounts vomited, and by an increased pulse rate of softer quality and usually with a pallor or beginning cyanosis. Often there is an apprehensive look.

These symptoms are due to intractantestinal stagnation and may be distinguished from those due to absorption from the peritoneal surface by noting the site of maximum intestinal distention. The reflex, purposive distention is most pronounced about the site of the lesion and usually corresponds to the region of greatest pain. Usually distention due to paralysis is progressive, and involves the whole abdomen simultaneously leading to vomiting after it has attained a certain degree of development. This distention is the result of the weakening of the gut wall due to a degeneration of the muscle coats while the purposive distention is reflex in character. The constitutional intoxication may be due to absorption from the peritoneum itself or from the contents of the paralyzed gut. That

it may be due to secondary changes in the gut contents is strongly suggested by a similar condition in intestinal obstruction in the absence of peritonitis. The studies of Draper suggest that the site of the genesis of the toxins is in the duodenum.

The suffering of the patient may be much augmented by the volume of the abdominal contents. So great may be the intra-abdominal distention that the general function of respiration is interfered with. This is usually abetted by lessened power of all the respiratory muscles. I have seen patients, particularly in childbed fever, who seemed to have their lives pressed out by the ever increasing abdominal distention. The last scene resembles that of mediastinal tumor.

It is instructive to watch the sufferers from dynamic ileus vomit their lives out. They vomit a large amount and then lie quiet for a time. Suddenly, apparently without warning, they expel another large amount. This vomiting seems to be an expression of gastric overloading from contents brought back from the small gut below by a process of reversed peristalsis. It differs from the primary reflex vomiting in that it appears as a welling up of large quantities of fluid, expelled without force. At this time peristaltic waves may be made out.

It may be differentiated from primary dilatation of the stomach by the absence of marked distention of the stomach, and from obstructive ileus by the manner of its onset.

Being a mechanical problem it can be solved only by mechanical means, namely by bringing about a state in which the gut can rid itself of its poison-laden contents with the least effort. This implies an enterotomy and it must be done before peristalsis ceases. Once the stage of reversed peristalsis is passed an opening in the gut does not drain, for, once the power of peristalsis has been lost, the gut collapses a short distance from the opening and prevents fluid from a greater distance from reaching the opening. This can be demonstrated in animals. The animal may breathe while the gut tract is entirely dead. Drainage to be of avail, therefore, must be made before the stage of paralysis is reached. Often the impending paralysis is presaged by the changed general conditions noted above, namely, the changes in the pulse and cutaneous circulation. When these appear action is demanded, for they in-

dicate that the process is beginning. The first part of the gut to suffer is usually the terminal ileum.

Fortunately the entire gut tract does not reach the same state simultaneously. The ileum may be paralyzed and dilated while the jejunum may retain its power of contraction. As a matter of fact, reversed peristalsis is often an expression of ileal paralysis with retained power of contraction in the jejunum. This reversed peristalsis must in a measure be purposive. If an obstruction is produced between two ligatures in a segment of gut in the ileum, reversed peristalsis may take place in the jejunum before the retained contents in the obstructed portion are allowed to reach it.

The relation of jejunum and ileum is important in indicating the site where the drainage should be made. The jejunum is the portion of the gut which retains the motive power longest.

The reason the ileum is usually the portion of the gut first to lose its motive power is that in the majority of cases the site of most intense inflammation is in this region of the abdomen. This is in harmony with the expressed belief that the gut paralysis is a toxic degeneration of the muscle cells themselves.

Even in those conditions in which the site of infection is in the upper belly, as in the perforation of ulcers, the great omentum acting as a water-shed conveys the infective material to the lower portion of the abdomen.

The unburdening of the gut tract by drainage may be accomplished by the formation of a permanent fistula or by incision with closure after the contents of the gut have been allowed to escape. McCosh advised drainage with immediate closure if after evacuation there is any difficulty in returning the intestines to their normal habitat. His procedure was actuated more by mechanical expediency for the convenience of the operator, than for relieving the gut. Lund deliberately planned the procedure with the purpose in mind of unburdening the intestines. He advises an incision at the obviously most distended portion; complete evacuation of the gut, then suture. This plan of incision, drainage and immediate closure of the gut is sufficient only in lesser degrees of paresis. At all times it has the disadvantage of soiling the operative area and if the gut is dropped back an infection and secondary fistula formation is always a possibility. When prolonged drainage is

necessary he advises suturing the most distended loop of the gut to the aponeurosis with the immediate introduction of a large catheter or glass tube. Heidenhain on the contrary failed to secure a ready flow so long as there was a tube in the gut. I believe both methods may be combined. In the first hours the tube serves to convey the fluid away from the abdominal wound. When the initial flow ceases the tube may be removed.

Lund believes the small gut is a more favorable site for the establishment of a permanent fistula than is the cecum, for in the event of its subsequent closure being necessary the operation is more simple here than if the fistula opens into the cecum. Lennander believes that when a fistula is made in the small gut at some distance from the cecum that portion of the gut lying between the fistula and the cecum collapses and becomes involved in a mass of adhesions making separation impossible and leading to an ileo-cecostomy. These opinions take into account only technical expediency, overlooking entirely the more weighty problems above noted. An opening in the cecum is permissible only when it is the terminal ileum that has lost its function. This usually represents a type in which a mechanical obstruction has resulted from adhesion of the gut to the site of inflammation. This type will be discussed under a separate heading.

Theoretically, of course, the best point for drainage is as near the cecum as possible so that as great an extent as possible of the gut will be emptied when the gut assumes its peristaltic function. A distended portion of the gut still active must be selected. If the opening be made in completely inactive guts the drainage will fail, for a collapsed area of gut lying between the ostium and the active gut will prevent drainage. Whether or not a gut is still capable of contraction is not easy to decide. Very often the extravasation of red cells from the vessels is marked by the presence of dark lines transverse to the long axis of the gut. These do not disappear on pressure and when present indicate that the portion of the gut is very seriously affected. Again when the gut is so distended that the wall is well nigh transparent one may be assured that it possessed but little power of contraction. In the presence of a deep red color which disappears on pressure, particularly if one can note some contraction of the gut wall between the fin-

gers, one may feel sure that this segment is capable of peristaltic action.

The portion of the gut in hand may be approximately judged by the rules discussed in the chapter on anatomy.

When it is possible to drain at the cecum Lennander's recommendations of the formation of a permanent fecal fistula at the cecum may be followed. Allaben and Reed likewise recommend this method. This site is selected by the last named author because it is believed to permit the introduction of fluids in the region where they will most readily be absorbed, quite overlooking the fundamental problem.

My experience has been that to be of value enterotomy must be done at the time of the primary operation. Done after this, there is an increase of the distention which results in paralysis, after this drainage has been uniformly useless in my hands.

As a prophylactic measure against impending paralytic ileus the procedure recommended by Kanavel may be followed. He advocates continuous gastric lavage coincident with continuous hypodermoclysis. Here if anywhere adrenalin should have its place.

*Obstructive Ileus.*—In some instances an actual intestinal obstruction exists in association with a peritonitis at the time of operation, or it may develop after the operation has been done. Not infrequently loops of small gut are so attached to an inflamed gut that an actual occlusion of the lumen occurs. After the appendix has been removed a loop of gut may become adherent in such a manner as to produce an occlusion. These conditions do occur, but they are diagnosticated more often than they occur. If an adherent gut is widely distended above the point of attachment and collapsed below, an obstruction exists. If distended above and below the point of attachment an obstruction does not exist. The recognition of an ileus is important, but meddlesome loosening of loops engaged in the beneficent mission of limiting the spread of infection is meddlesome surgery. Such adhesions are often loosened with the idea that they will remain and cause trouble later. There is no such danger. These inflammatory adhesions always loosen.

The recognition of an obstructive ileus is dependent on the dem-

onstration of stercoraceous vomiting with the absence of clinical symptoms that would accompany a dynamic ileus.

When an obstructive ileus exists the lumen must be made patent. Sometimes it is easy to loosen an adhesion. If there is an inflammatory mass involving a convolution of guts, it is best to do an enterotomy, awaiting the natural course of the disease to loosen the adhesions. Possibly a resection may be necessary later but if so a more favorable time can be selected.

**Drainage of Extraperitoneal Abscesses.**—Abscesses strictly extraperitoneal are those which lie outside the parietal peritoneum. In a clinical sense walled-off abscesses may be considered as being extraperitoneal since they are such so far as their relation to the free peritoneal cavity goes.



Fig. 164.—Abscess within the broad ligament drained through the vagina.

Surgeons generally recognize the importance of avoiding the peritoneal cavity when draining the extraperitoneal spaces, except in abscesses of the pelvis, particularly in those of the broad ligament. Many operators seek to drain these abscesses by abdominal section. The difficulty is perhaps less the failure to comprehend the general principle than one of mistaken diagnosis. Broad ligament abscesses are mistaken for pus tubes.

Broad ligament abscesses are the result of infection of the lower segment of the uterine tract, hence the abscess has its chief location at the base of the broad ligament. These are best drained by opening lateral to the cervix, care being taken not to penetrate the overlying peritoneum. It is only the minority of broad ligament

indurations that break down to definite abscess formation hence solid masses are often cut into. If pus is not readily found it is best not to continue the search too long lest a gut be perforated.

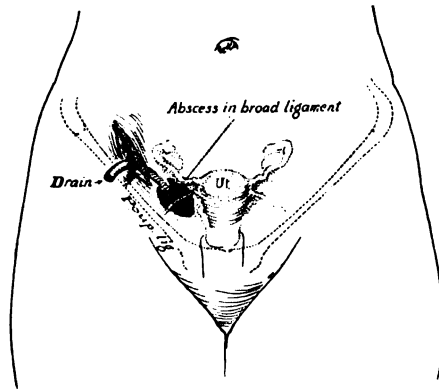


Fig. 165.—Abscess situated far laterally in the broad ligament drained by an incision above Poupart's ligament. (Drawing modified by Cullen.)

An isolated pocket may find the opening or at least the incision may deplete the indurated area, thus hastening the resolution.

When the infected area lies over the body of the ischium, it may

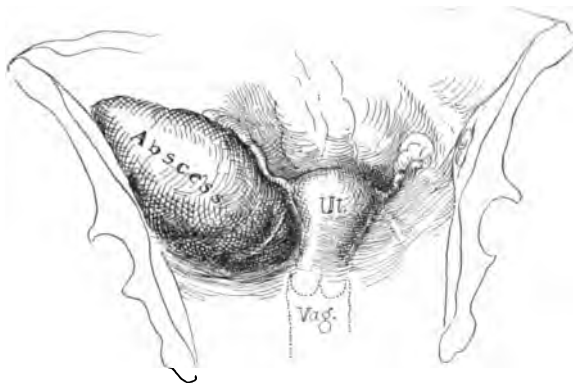


Fig. 166.—Large broad ligament abscess pointing both in the vagina and over Poupart's ligament. These are best drained above if the infection is mild.

be reached with difficulty from below. In such instances it may be more readily reached by making the incision over the medial half of Poupart's ligament and by lifting the peritoneum approach



the infected area. When these abscesses become large they point above Poupart's ligament (Fig. 166), and may then be easily reached as indicated above. Sometimes low pelvic infections are

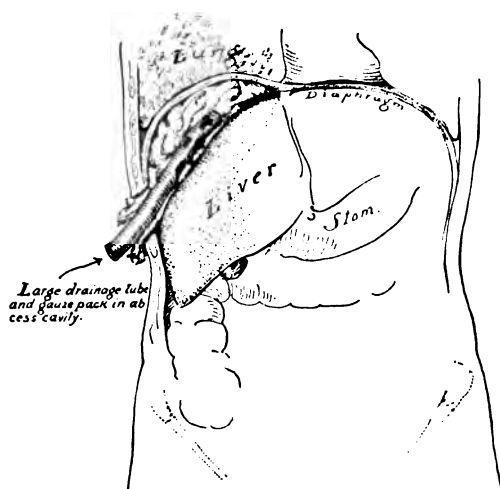


Fig. 167.—Drainage of subdiaphragmatic abscess below the costal margin.

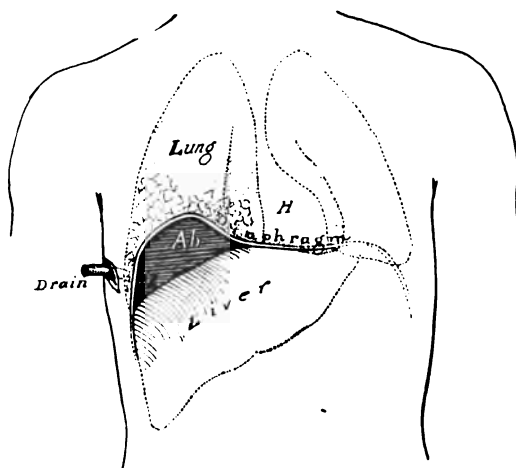


Fig. 168.—Subdiaphragmatic abscess drained transpleurally after the pleural space had been obliterated by packing it a week with gauze.

accompanied by suppuration of the upper group of inguinal lymph glands. These must not be mistaken for the extension of infections continuous from the pelvis. Perirectal abscesses of course

are reached by perianal incision. Abscesses high in the sigmoid form abscesses about the rectum, sometimes of considerable magnitude. When fluctuation from the rectum can be palpated, an opening into the gut can be made. Sometimes a pararectal incision will suffice. At any rate transperitoneal drainage should be avoided.

Extraperitoneal abscesses may result from appendicitis. This may occur when the appendix is anatomically extraperitoneal or when walled in by previous adhesions. These are readily drained by an incision along the edge of the quadratus from the twelfth rib to the crest of the ileum. These abscesses are treacherous, for they tend to extend upward to between the liver and diaphragm in Morris's pouch, or into the lung. When there is an extracolonic infection and the patient has pain on deep breathing these spaces should be investigated. Once the infection gains the space between the liver and diaphragm it is little likely to become limited before the coronary ligament is reached. The reason for the failure of limitation is that there is no subperitoneal connective tissue, hence adhesion forming reactions can not take place. In such cases free drainage should be made between the liver and diaphragm (Figs. 167, 168.)

Infections about the duodenum can be more safely drained from the side than from the front. When so approached at least one wall of the drainage tract does not endanger the peritoneum.

When the pancreas requires drainage an opening from behind is made, but unfortunately only the knowledge gained from a median laparotomy enables the surgeon to make a diagnosis. Abscesses from slowly perforating gastric and duodenal ulcers must be drained transperitoneally. When there is an adhesion to the anterior abdominal wall the abscess may be entered without opening the peritoneal cavity. When it is discovered after opening the abdomen that the abscess is walled off but that the peritoneal cavity is free above, it is safest either to pack about the abscess and await until adhesions have formed before opening into the abscess or to seek to reach the abscess from some route which will not traverse the peritoneal cavity.

In abscess of the solid parenchymatous organs the same plan may be followed.

In all such instances an abundant packing off with gauze should

be practiced. The chief service of such a gauze pack is to excite a rapid walling off of the region represented by the gauze.

Infections resulting from infections of the soft parts which approach the peritoneum may menace it in more instances if the diagnosis is mistaken. Infective foci in bones or in muscles may simulate intraperitoneal abscess and the unwary surgeon may seek to open in transperitoneally.

**Drainage of Intraperitoneal Walled-off Abscesses.**—An abscess which has been formed by the common adhesion of abdominal viscera must be drained by some route that does not traverse any part of the free peritoneal cavity.

When, as in an appendicitis, the abscess is walled off, there are commonly adhesions to the anterior parietal wall. Usually an incision placed widely lateral will reach the abscess without traversing the free peritoneal cavity. When the abscess lies more medial the peritoneum may be lifted from above Poupart's ligament and the abscess approached extraperitoneally from behind.

When an abscess has formed in the pelvis the result of adhesion of gut coils and omentum about the appendix, the peritoneum may be pushed from the pubis until the bottom of the pelvis is reached and then the abscess opened from below. When the abscess is large it may be opened into the rectum. Unless the abscess is well defined this procedure may result in inadvertently opening into the gut. In parous females the drainage can be most conveniently made, of course, through the vagina.

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## CHAPTER XVIII

### OPERATIONS ON THE PERITONEUM

**General Principles of Peritoneal Sutures.**—The fundamental factors involved in the suture of the peritoneum were discussed in the chapter on wound healing. It is necessary here only to consider those factors directly involved in technic, and to consider such refinements only as are practicable in every-day surgery.

In the surfaces it is desired to unite sufficient irritation is produced by the injury of the needle and from the pressure of the suture to excite a plastic exudate. It is necessary that this coaptation be maintained for a time sufficient for the formation of fibrous tissue—at least two days. If the suture holds less than this, if traction of a sufficient degree is applied, the surfaces may separate. When there is a question of ideal asepsis, as in making anastomoses, this period should be lengthened fourfold. When in conjunction with coaptation a sufficient pressure is produced to check or embarrass the circulation, or where vessels of some magnitude are accidentally or designedly included in the suture additional disturbances may be produced which may make a more permanent suture desirable. When the peritoneum is severed as in most operations the cut edge in addition furnishes an abundant plastic exudate. Simple puncture and ordinary pressure is not sufficient in plications, as in the shortening of the round ligaments to produce adhesion of the deeper structures. Here the structures beneath must be exposed to direct union or if the peritoneum is included pressure must be made by a sufficiently permanent suture so that the peritoneum will be destroyed and thus permit the supporting structure to come in apposition secondarily.

The requirements of technic involving the peritoneum are considerably more diversified than is required in the skin. In the latter simple coaptation is all that is required. In the peritoneum the coaptation must be all that a skin suture needs to be and often much more. In parietal incision the only requirement is a coapta-



tion sufficient to exclude adhesion to mobile intraabdominal organs and resembles in principles a simple skin suture. In cases where anastomoses of hollow organs are made the suture must be more secure in order to prevent the escape of the contents of the viscus. When adhesions are to be severed yet another factor enters.

*Material Used.*—Material suitable for suturing peritoneal surfaces must be of sufficient durability to permit healing, as above indicated, under the varying conditions found in the abdomen, and it must be sterilizable. Two general classes only need be considered, the absorbable and the permanent. Of the former catgut alone is to be considered, while in the latter class silk and linen must receive consideration.

*Catgut.*—As now prepared catgut is sterile. The question only arises as to the best manner of preparation for the use in the peritoneum. Plain catgut is too ephemeral to warrant its use in the peritoneum. It lasts at most not more than two or three days and even more quickly than this it becomes so attenuated that it no longer acts as an efficient coapting agent. Of the various hardening processes those giving the least irritant to the surrounding tissues are preferable. The pyocetanin guts are little irritating and in situations where the wound receives other support as when fascia is closed over it or where flaps as after adhesions are to be united, is efficient. Chromic gut is more irritating but more durable and in the smaller sizes 0 or 00 makes a desirable material for peritoneal plastic operations. Iodized gut because of its irritating action tends to produce wide adhesions and is not to be considered. Many surgeons use catgut throughout for anastomoses of hollow viscera and if prepared so that it remains the necessary length of time it no doubt is the ideal suture.

*Linen.*—Linen especially when impregnated with celluloid, makes a desirable material. It is more pleasant to use than silk but can not be had in sizes as fine as should be employed. It should not be reboiled because it tends to become fragile.

*Silk.*—Silk makes the most desirable material for routine use and can be had in fine sizes. It is strong and is readily sterilized if sufficient care is used. One objection to silk is that it does not slip readily through the tissues. This objection is overcome in great measure by not using unnecessarily long strands and by pre-

venting any kinking. Another objection to the general use of silk is the likelihood of its becoming infected. Kocher has well said that to use silk successfully one must be a master of aseptic technic. The chief objection is that it remains too long in the tissues. When a silk suture is used it becomes covered by a plastic exudate. This exudate forms the new peritoneum over the line of suture and covers in the permanent suture. The tissue within the grasp of the ligature becomes destroyed by a process of pressure necrosis and gradually works its way into the lumen of the gut. This means that an infected tract must extend to the suture. When a running suture is employed this extrusion of the ligature requires a considerable length of time, at least a number of weeks, and during this process healing can not be completed. This invites an ultimate extended cicatricial ring but chief of all is that an extensive induration may take place about the line of suture which may form a thick mass resembling a carcinoma in feel and may be so extensive as to occlude the anastomotic ostium. I have lost two patients from this cause. There is another objection hitherto not recorded so far as I know. The minute abscesses about the silk admits bacteria to the general circulation and distressing arthritides may begin at the time this process is active. These clear up in time but are the subject of intense distress during this progress.

Though the classes above noted involve the same underlying principle above discussed, the differences in technic are such that they must receive separate consideration.

**The Closure of Peritoneal Incisions.**—The fundamental problem in the closure in any incision through the peritoneum is to secure coaptation of surfaces and prevent the severed edges from presenting within the abdomen and thus to invite adhesions with the omentum or other structures. This end is best assured by inverting the edges so that the severed edge will present externally into the wound and become buried between the muscle layers when these are coapted (Fig. 169). This may be accomplished by a Lembert or mattress stitch done from the opposite surface from that from which it is usually done. The suture must be firm enough to prevent gapping when the elasticity of the elastic layer has been in play for some time. The importance of this can be readily determined by opening the abdomen of animals after varying intervals

following the operation. One will be astonished at the imperfect coaption revealed after a day or two. Because of the elasticity of the subsera and the disposition of the catgut to elongate when softened by the tissues the line of union relaxed and raw edges be-

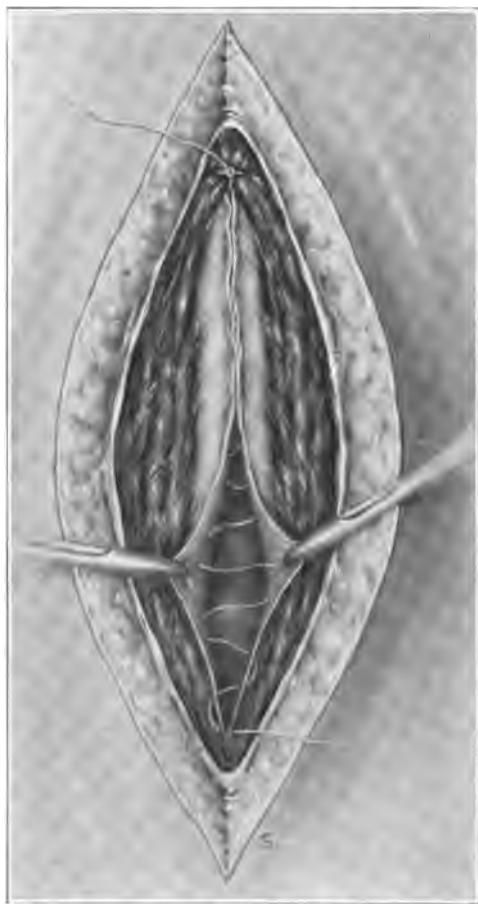


Fig. 169.—The suture begins by including the recti muscles. A running suture coapts the peritoneal surfaces, everting the edges between the muscle layers.

come inverted into the abdominal cavity inviting adhesions to surrounding viscera.

In order to prevent this inversion the suture must be so placed that the edge is held up by the entire line of suture. When so

applied the cut edge can not become everted unless the suture gives way. This result can not be secured when the usual over-and-over running suture is employed.

The suture best employed is one that retains its tensile strength for five or six days. Silk in small sizes would be the ideal suture were it not that it remains permanently in the tissues and should infection occur would be a source of great annoyance. The more ephemeral forms of catgut are objectionable because they may disappear too early. Midway between these two is pyoctanin or chromic catgut. The smallest sizes are sufficiently strong and cause less irritation than the larger ones. No. 0 pyoctanin or No. 00 ten-day chromic are the most satisfactory sizes.

Because of the elasticity of the peritoneum in the lower median incision an uninterrupted (continuous) suture retracts the ends of the incision producing a puckered rather than a linear one and if this retraction continues after the suture is tied, relaxation of the suture line takes place permitting the cut edges to reach the intraperitoneal cavity even though the Lembert suture has been used. Were it not that the interrupted suture requires more time this would be the ideal suture. Some of the advantages of the interrupted suture may be obtained without undue loss of time by back-stitching, which fixes the suture every few centimeters. Relaxation may be further prevented by fastening the suture to the overlying fascia at either end of the incision. This may be accomplished by passing the suture through the muscle or fascia or by tying in with the sutures of the overlying areas (Fig. 170). This maneuver has the additional advantage of preventing the formation of a dead space in the region of the pubes.

In those regions where the peritoneum is very intimately attached to the fascia and there is much tension as in the upper abdomen of fat persons, a staple or mattress suture including both fascia and peritoneum is often the best. A medium-sized chromic gut of greater durability should be employed when this plan is followed.

**Suture in Hollow Viscera.**—In the suture of hollow viscera whether for the repair of wounds or for the formation of permanent ostia between neighboring organs, the principles are the same. In addition to the general principles laid down for parietal sutures

here several factors are added. While in parietal sutures close coaptation of the edges is desirable here it is imperative lest the general peritoneal cavity become infected from material within the organs sutured. Fortunately the environment is such that the



Fig. 170.—After the entire peritoneum has been closed as in Fig. 169, the same suture continues back, coapting the muscles to the point of beginning.

more permanent sutures may be used without objection. In addition a protective line of sutures is usually employed.

This preliminary line was first used by Czerny and bears his name. It may include the cut edge of the mucosa only, the mucosa

and muscularis or each of these layers may be united by a separate line of sutures. Aside from protecting the peritoneal sutures from infection they serve as hemostatic sutures as well.

The exact method of securing apposition of the peritoneal surfaces is immaterial. The classic method is that first employed by Lembert. This and the mattress sutures introduced by Cushing are those most generally employed.

While continuous sutures are now most generally used because of convenience, the interrupted suture is theoretically most correct. Running sutures all have the objection that as the tissues relax the sutures become correspondingly less secure. Fortunately the reactive infiltration of the tissues usually is sufficient to take up this slack. If long lines of continuous sutures are employed the line should be secured by interlocking sutures.

While the Lembert type of sutures is said to include only the peritoneal layer, the peritoneal suture is too delicate to withstand possible traction. For this reason the suture must be deep enough to include the submucosa as well, as practiced by Halsted, in order to secure the firm attachment offered by this resistant tissue.

Because of the proximity of this suture line to the lumen of the hollow viscera a new problem presents itself. The gut contents may quickly absorb a catgut suture making the suture line insecure in a surprisingly short time. The softer catgut will disappear from the stomach or upper gut wall in a day or two (in dogs) exposing the deeper sutures to the action of the intestinal juices. The harder varieties of gut withstand digestion for a number of days, usually long enough to permit the peritoneal apposition to advance well along to fibrous union.

The outer layer of sutures, that apposing the peritoneal sutures, is the one depended on to prevent leakage while holding the apposed surface in a position calculated to secure the most certain union. If all goes well and there is no interference with healing, catgut will maintain this apposition for a sufficient length of time. Should any factor develop which delays this union the gut may disappear before healing is sufficiently advanced to prevent leakage. Because of this possibility most operators prefer silk for this layer. Nor is there any objection to the use of this material such as was advanced against its employment in the parietal peritoneum.

The reason for this is that the silk does not remain permanently imbedded in the tissues, but is extruded into the lumen of the gut and is thrown off. This expulsion may take place in an astonishingly short time, often as early as 10 days after the suture is placed.

Theoretically it should be possible for silk to become imbedded in the line of suture. This would demand that they be placed in an aseptic field. This obviously is not possible in such close proximity to the lumen of the gut. At any rate I have sectioned many



Fig. 171.—Adhesions between sigmoid and broad ligament and between rectum and uterus. There is an intraligamentous cyst on the right side.

guts that were sutured and have never discovered any imbedded silk. Even when two gut surfaces are united without making opening into the gut the silk is expelled into the gut.

**Suture of Adhesions.**—The general principles here are the same as those indicated for incision in the parietal peritoneum—the cut edges must be everted from the general peritoneal cavity. Here the problem is concerned chiefly with securing sufficient tissue to cover the denuded area or to the gaining access to the region requiring suture. Where

there are broad surfaces to unite frequently sufficient tissue is not always available. As already noted in the section in the prevention of adhesions, this difficulty may in a measure be anticipated by cutting the adhesions with the same deliberate planning as is exercised in making a skin flap in amputations (Figs. 171, 172, and 173). It is comparable to an amputation in which both proximal and distal stumps are covered with skin. Such adhesions are often seen about the gall bladder and particularly about the ovaries and tubes.

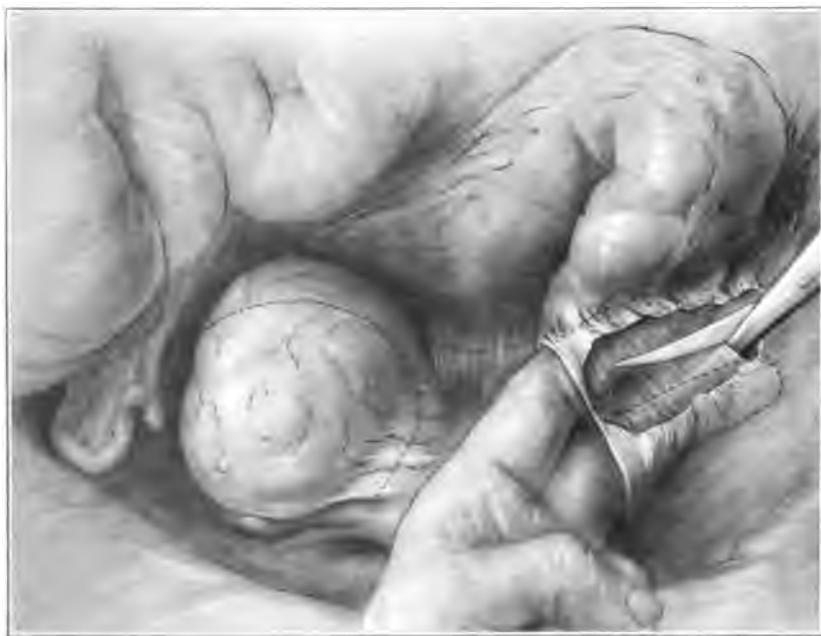


Fig. 172.—The adhesions in Fig. 171 are so incised as to permit a covering of the denuded area after the operation is completed.

The difficulty in such cases is that the peritoneum when cut tends to retract so that difficulty is experienced in finding the edges one wants to unite. This predicament may be obviated by fastening these edges with forceps as they are cut. They can then be readily identified when it comes time to apply the sutures.

When there is a single plane of tissue as when the omentum is attached to some surface it is best managed by rolling the cut edge



in the folds of the peritoneum and fastening it there with sutures. If the adhesion contains large vessels, as in the omentum, they must be first ligated.

**Covering by Transplant.**—When two surfaces are broadly attached there may not be sufficient peritoneum to cover either or both surfaces. If it is possible to cover but one surface, when possible that surface is elected for repair which demands the greatest degree of mobility in performing its function. For instance, when



Fig. 173.—The adhesions severed in Fig. 172 have been united by Lembert sutures.

a gut is attached to the parietal peritoneum sufficient parietal peritoneum is dissected off to cover the denuded area. The surface so denuded on the parietal wall may be covered by mobilizing peritoneum in the immediate vicinity, or from a distance. The same problem offers itself when there are gut adhesions to the broad ligament. Sometimes there is not sufficient peritoneum available to cover either denuded surface as when a loop of gut is broadly attached to the uterus.

In such an event when the denuded area is too extensive to be

closed by direct suture over the gut surface a flap may be formed from one leaf of the mesentery according to a method developed by Richardson. This same plan may be employed even more effectively in case of the ascending or descending colon by mobilizing the parietal peritoneum.

The most obvious tissue available for the repair of defects in the peritoneum is the great omentum. Senn first employed this structure for this purpose. His plan was to draw a convenient tip



Fig. 174.—The peritoneum is incised separately in order to secure the necessary tissue for covering the denuded area.

of omentum to the region involved and fix it there with sutures. The use of this method is made more or less hazardous by the fact that organs may be unduly fixed or loops of gut may become entangled in the loop of omentum so formed. This method is applicable only when the denuded area is somewhere near the root of the omentum. One would hardly care to risk its use for instance on the fundus of the uterus.

The use of the omentum in this manner is particularly desirable where there is disturbance of nutrition of the organ whose surface is denuded. On the whole omental grafts have but a limited use in the prevention of adhesions.

The use of detached omental grafts would seem to avoid the objections above enumerated and yet make an ideal covering. But a new difficulty arises: A detached bit of omentum partakes of the reaction of the environment to which it is carried and by add-

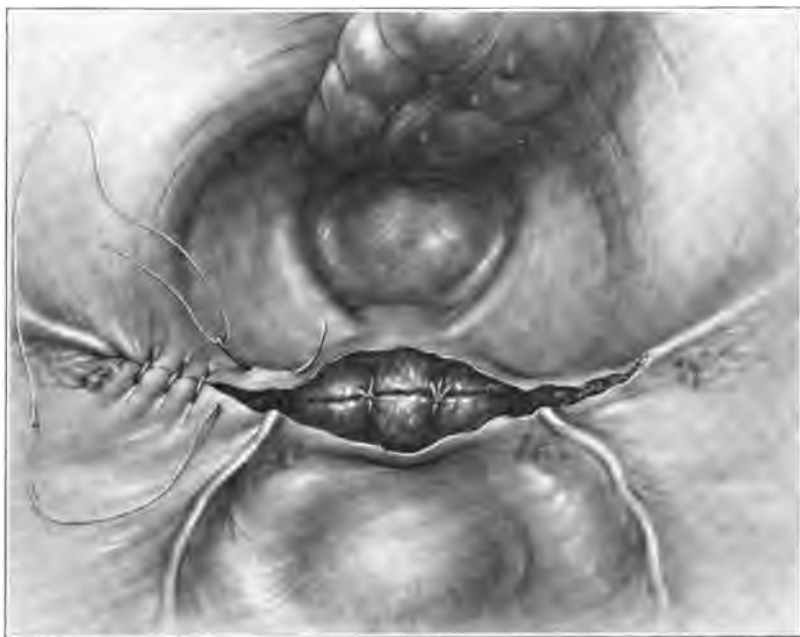


Fig. 175.—The flap planned in Fig. 174 on being closed.

ing to the exudate actually aids in inciting adhesions. It is only when a denuded surface is likely to be particularly pernicious that one is justified in making an omental transplant.

**The Removal of Extraperitoneal Organs.**—When organs lying extraperitoneally are to be removed transperitoneally it is desirable to plan the flaps to cover all denuded areas before the operation is begun. By so planning the entire operative field may be completely covered and adhesions most certainly avoided. The

removal of tumors, and particularly of the uterus and hysterectomy presents the most frequent opportunity to exercise this technic (Figs. 174 and 175).

**The Suturing of Solid Viscera.**—The solid parenchymatous organs require suturing when injured. Because of the delicacy of the peritoneum in this situation it offers little resistance to the suture lines. For this reason suture material of large diameter is employed and often this is reinforced by placing some foreign material on the surface of the organ in order to keep the suture from cutting through. Magnesium plates or gauze are most frequently used for this purpose. Because of the density of the subperitoneal tissue inversion of the edges of the peritoneum is never possible and the surgeon must be satisfied with simple coaptation.

## PART II

### CHAPTER XIX

#### APPENDICITIS

##### APPENDICEAL PERITONITIS

Affections of the appendix serve as the most frequent starting point of peritonitis. About this small organ the entire chapter of etiology and pathology, if we knew enough, could be written. Despite the vast amount of literature that has been written, our knowledge is still very elementary from both a theoretic and a practical standpoint.

**Historical.**—Between the time of Mélier and Fitz, a period of some fifty years, a large number of papers appeared which exhibit a long series of side-stepping hardly paralleled in medicine. Numerous papers appeared which seemed clearly demonstrative of the truth, but the rank and file of the profession remained oblivious to their significance. A brief mention of a few of these may be permitted.

Mélier was the first man to make a general statement as to the probable frequency of disease arising in the appendix. His idea of the pathogenesis was that the appendix became gradually distended with fecal matter until it became inflamed and gangrenous. He records five cases. He correctly interpreted the recurring type, and even suggested the possibility of operative treatment. It seems that the clear presentation of this author would have led to a ready acceptance of the facts. In this instance, as so often happens in medicine, progress was blocked by one of the ablest men of the time. Dupuytren would have nothing of the new idea. He insisted that the peculiar conformation of the intestine in the right iliac fossa, and the fact that the intestinal contents changed in their character, accounted for the frequent abscesses in this

region. Then followed a long period of floundering. Goldbeck introduced the term *perityphlitis*, about which so much contention followed. The clinical symptoms of localized tumor and abscess were admirably worked out, but little appeared which shed light on the true nature of the disease. Bright and Addison noted that the appendix was often found in these abscesses, and that its extremity was often perforated; but because the cecum was itself inflamed they concluded that the appendix had little to do with the origin of the disease. They correctly noted, however, the importance of fecal concretions in the appendix. These authors possessed an astonishingly clear conception of the general principles of treatment. He drew a parallel between a broken leg and the inflamed peritoneum. He notes that to compel the fracture patient to attempt to walk would be as reasonable as to force a bowel movement in peritonitis. He insisted that perityphlitis was secondary to inflammations of the appendix. He was the first to note that the disease may terminate without suppuration. Lewis tabulated forty-seven cases. He notes the variation in size of the lumen, and the possibility of an obliterating inflammation occurring. Even so early in the history of the disease appendiceal involvement was already exaggerated. In 300 autopsies With found the appendix diseased in 110. His paper emphasized the importance of the appendix as the etiologic factor in appendicitis. The papers already quoted furnish a suitable background for the contribution that finally awakened the profession. Fitz, in a series of papers, emphasized the fact that the cecum was intact while the appendix was ulcerated and perforated. These statements showed clearly that the old term "perityphlitis" was misleading. But he did more. He introduced the new name *appendicitis*, which, though a hybrid, served to fix the attention upon the organ at fault by means of two terms very easily comprehended by the profession. It must be noted that the impression these papers made on the profession was due in part to the readiness of American surgeons to put into practice the ideas of treatment suggested by Fitz. It was also Fitz who first noted the true relation of the appendix to abdominal infections, and presented the fundamental dictum that peritoneal inflammations could be dealt with successfully only when they were localized. This made it imperative that the point of depar-

ture of such inflammations and the study of their cardinal symptoms be developed to the utmost.

Following Fitz came a stupendous mass of literature which served gradually to clarify both the pathology and the symptomatology of appendicitis. The important part of this literature is so recent that the proper place for its consideration is in the review of our present clinical resources. History here merges into current events.

The few milestones here indicated serve only as a starting point for the student of history. More extended presentations will be found in Kelly and Sprengel. The former contains a select, the latter a complete bibliography.

### **Etiology**

The etiology of appendicitis concerns us here only in so far as it involves the peritoneum. Nearly all affections of this organ do involve its peritoneal covering; and its inflammations attain clinical dignity only because they cause gross changes about it. The whole range of appendicitis falls almost entirely, therefore, within the realm of peritonitis. Certainly there is no other process which so often gives rise to peritonitis. Therefore, from the practical standpoint the appendix is the very center of interest to the student of peritonitis. A somewhat careful study of the few facts known relative to etiology helps materially in comprehending the pathogenesis.

**Heredity.**—Appendicitis like glandular diseases seems to have a predilection for certain families. Forschheimer records a family of fifty-two members, representing three generations, of whom 17 per cent had appendicitis, and another in which five out of twenty-five members were affected. Giertz mentioned numerous instances in which a parent and two children were affected. Albarran reports four cases in one family, and Treves cites one family in which there were five cases. I can duplicate in my own experience the number of cases of each of the last two writers. I have had one family in which a sister and four brothers were operated on by me in the acute attack; and another brother has had three attacks, but has not been operated on. In another family, four were oper-

ated on by me in the acute attack, and a fifth member was operated on by a colleague.

Families in which tonsillar disease is prevalent are more apt to be affected by appendicitis. Tuffier believed that a hereditary deformity of the appendix might play a part, and Delbert believed that a general digestive predisposition might be a predisposing factor.

**Age.**—Nearly all authors are agreed that appendicitis most often affects persons between the ages of ten and thirty years. Probably more than 50 per cent of cases occur in this period. Krogius, for instance, presents the following percentages as to ages: 0 to 10 years, 2 per cent; 11 to 30 years, 36 per cent; 31 to 40 years, 16 per cent; 41 to 60 years, 16 per cent; above 60 years, 8 per cent. These statistics give a greater percentage to middle and later life than comes to the experience of most surgeons. Nearer the average experience are Gullstadt's figures: below 15 years, 15 per cent; between 15 and 30 years, 57 per cent. Albu's statistics, on the contrary, give too low a percentage to advanced life. He found only 8 per cent after the age of 30 years. Nothnagel in the collected statistics of 954 cases had 306 between 11 and 20 years, and 323 between 21 and 30 years. Giertz gives statistics that seem to be representative. Of 533 cases, in 42 the patient was less than 10 years old; in 329 between 11 and 30 years; in 81 between 31 and 40 years; in 33 between 41 and 50 years; in 22 between 51 and 60 years; and in 6 above 60 years old. This series is particularly trustworthy because it includes only the cases in which suppuration occurred. In "interval" and "chronic" cases there is often uncertainty about the diagnosis, even after a microscopic examination has been made.

The statistics above quoted are sufficient to indicate the great preponderance in young adult life. Why young persons are most frequently attacked is not known. The most plausible theory is that at this age the entire lymphatic apparatus is more often involved in inflammatory affections, and since the appendix is made up largely of lymphoid tissue, it naturally shares the same fate. To say that the appendix is longer and larger in young persons proportionately is but restating the same thing.

**Sex.**—The general impression prevails that the male is more fre-



quently affected than the female. This is particularly true when the suppurative type is considered. Krogius, however, gives 27 per cent males to 28 per cent females. If careful histologic examination is made of appendices coming to any laboratory, I dare say a greater number removed from females will test the ingenuity of the pathologist to confirm the diagnosis. In my own cases, in persons over twelve years of age, 90 per cent of suppurating cases have been in males. In children below 12 years of age there has been a preponderance of girls. Many authors find a much less marked discrepancy. Hansen had 156 males to 105 females. Riedel had 955 males to 577 females. Of Albu's acute cases, 61 per cent were males. Giertz had 321 males to 212 females in the suppurative cases. In children, in his statistics, females predominated,—54 per cent to 46 per cent males.

The cause of preponderance in males has had no explanation save that in females a collateral circulation through the appendiculo-ovarian ligament exists. This is certainly an ingenuous explanation, since the existence of such a vascular connection between these organs is by no means certain in the vast majority of cases.

**Occupation.**—Giertz classifies his 533 cases according to employment. Of 311 patients who followed physical occupations 56 per cent were males and 44 per cent females. Of those not engaged in physical labor, 76.4 per cent were males and 24.6 per cent females.

**Diet.**—To meat diet has been ascribed an influence by many writers, notably by Lucas-Championniere, Kümmel, and Flesch. As evidence of the influence of meat on the causation of appendicitis its rarity among vegetarian peoples is noted. Thus Naab found this disease rare in the Turkish army, and Prölss found no cases in the natives of East Africa.

**Trauma.**—The relation of trauma to appendicitis formerly had but an academic interest. Since industrial accident insurance is becoming constantly more widespread trauma assumes a very vital practical interest, and it becomes necessary to form some general plan by which the problems presenting themselves may be solved.

Trauma that may incite an acute appendicitis may be caused by a blow from without; by striking the abdomen against some object; by a sudden jar, subjecting the abdominal contents to sudden violent movements, either by striking a remote part of the body,

as in falling on the feet or buttocks, or by a sudden increase of intraabdominal pressure, as in warding off a blow. Certain authors, notably Wätzold, include as traumatic causes those induced by the irritation of foreign bodies that have been swallowed. These are readily separated from the first class, since they obviously do not readily involve responsibility in industrial insurance. Copland was the first to report a case of appendicitis due to trauma. Whether or not it is possible to produce an appendicitis in a normal appendix by a blow is difficult to determine. Haist denies any influence. Nothnagel, Sonnenburg and Sprengel do not believe that it can do so, while Cassanello and Neumann believed that it might do so. In order that trauma shall be ascribed any etiologic importance the symptoms must begin in the first 48 hours, according to Sonnenburg, and Jeanbrau and Aglanda. Sonnenburg notes that there is no relation between the severity of the trauma and the extent of the disease. In collected statistics Giertz notes that in 10,888 cases of appendicitis trauma was operative in 410 cases. In his own statistics of 533 cases, trauma was assigned as the cause in only 4 cases. In the recorded statistics there is a wide variation between the estimate of Hawkins, with 54 per cent, and Ebner with only 8 per cent. Here again Sonnenburg's opinion is worth noting, since he was dealing with the problem in a country where industrial insurance had already long been in operation. Of 3,480 cases he ascribes an etiologic influence to trauma in 1.5 per cent of the cases.

It is necessary to differentiate cases in which the first symptoms of the disease immediately followed trauma from cases in which an existent disease was aggravated by trauma or a renewed attack apparently was precipitated. We may compare these cases with those in which a sclerosed artery of the brain gives way after a relatively slight traumatism.

In my own experience in only two cases was there an immediate onset of symptoms following trauma in patients previously free from the disease. One of these was in a girl of nine years struck by a thrown ball, the other in a boy of sixteen struck by a comrade's knee in a game of football. I have had numerous cases in which trauma was alleged to have been operative. The most of these cases either carried liability insurance or desired to secure

indemnity from a corporation by which they were employed. In most of these the very existence of trauma was not established. These factors complicate the problem, for it is well known that very extensive visceral lacerations may be produced by injuries that leave no external mark of injury. The manner of statement and the alleged character of the injury are the only guide in such instances. Among the more flagrant of the simulant cases are those in which local pain is the only evidence of the existence of appendicitis, with an absence of all the cardinal symptoms. If joined to this the individual is overanxious for surgical treatment he may safely be stamped as a malingerer.

The relation of trauma to recurrent attacks is problematic as a general proposition. Fink looks on them as mere coincidences. It is easy to conceive, however, that an appendix recently recovered from an attack might be more readily set into renewed activity than would a normal appendix. The nature of the trauma and the time of onset may throw some light on the probability, though in general the surgeon will be obliged to accept the patient's statement, at least as to the existence of trauma. In those instances in which there is a fecal concretion in the appendix trauma may be conceived as being more likely to stimulate mischief than one in which there is no foreign body.

The question of the exaggeration of an existing appendicitis by trauma is more clear. I have seen several such cases. In one case a man, aged 46, who had successfully passed through the localizing process was thrown to the floor by the breaking of the cot on which he lay. The effort to rescue himself was immediately followed by a sharp pain in the abdomen. Laparotomy, undertaken twelve hours later, failed to check a rapidly extending peritonitis. In another instance the transportation of a patient a considerable distance soon after apparent recovery, was followed by abscess formation. This may have occurred under rest in bed, but renewed pain set in during the journey. I have seen rapid extension follow manipulation by a too enthusiastic osteopath in two cases.

Appendicitis in an irreducible hernia is not an uncommon occurrence. I saw one such case in which a suppurative appendix was associated with volvulus of the cecum in an irreducible hernia. In such cases the additional tax on the circulation produced by

the displacement may well be considered as making the organ more vulnerable to injury.

**Fecal Concretions.**—No other factor in etiology presents so much tangible evidence as inspissated fecal masses in the appendix. In the perforative type particularly, it is common to find such a concretion free in the peritoneal cavity or yet remaining in the lumen of the appendix. A study of this condition gives us a better notion of the genesis of general peritonitis than any other. In 400 autopsies Ribbert found concretions in 10 per cent, and in only one of them was there a perforation.

Concretions are found relatively with much greater frequency in acute suppurative or perforative appendicitis than in the simpler types. Sprengel has collected a large series of cases, and fecal concretions were found in about 40 per cent of them. Among these may be noted Fitz's 100 cases, in 47 of which concretions were found, and Murphy's 30 per cent in 141 cases. The association is much more constant in the perforative than in the simple cases. The conclusion drawn by Sprengel is that those in which there is a concretion are much more liable to perforation, and it may be added, are much more apt to be followed by an unlimited peritonitis. Whether or not previous simple attacks predispose to the formation of fecal concretions is a question which, it seems to me, may receive an affirmative answer. In most cases in which a fecal concretion is discovered a history of previous attacks may be obtained. Since, according to Sprengel, in 85 per cent of the perforative type a concretion is found, the conclusion would seem warranted that the concretion was formed subsequent to a mild attack of appendicitis. This may account for the relatively less common occurrence of concretions at the present time, assuming that not a sufficient number of attacks to develop a concretion are allowed to take place before the organ is removed.

**Foreign Bodies.**—In the early period of the study of appendicitis foreign bodies were frequently described, due to mistaking fecal concretions for cherry and grape or other seeds. Other foreign bodies have repeatedly been noted, such as pins, fishbones, shot, and small bullets. Mitchell has made a collective report of such cases, and Kelly has extended the list. Dawbarn and others have reported a number of cases in which fishbones were found. In-

testinal parasites have been noted. Sprengel has published a list of such reports.

**General Infections.**—The association of acute appendicitis and other infections has often been observed by surgeons. A tonsillitis followed in a few days by appendicitis is of relatively common occurrence.

The relation of rheumatism and appendicitis is less generally recognized. Finney reported three cases in which rheumatism was associated with appendicitis. In the light of our present knowledge it would probably be more nearly correct to speak of infective arthritis associated with appendicitis. I have seen a scarlatiniform eruption follow appendicitis in which there was a septic mesenteric thrombosis. The frequent association of gall-bladder disease has been assumed in many cases but less often proved.

La grippe likewise may precede appendicitis. To accept merely a clinical diagnosis, however, I believe is unwarranted, since not infrequently in this disease many of the early symptoms correspond to those of appendicitis, but the subsequent course makes such an involvement unlikely. The demonstration of an actual disease of the appendix during an attack of la grippe may be taken as an indication of relationship, but of course the actual demonstration of the influenza bacillus is required for final proof.

### Pathogenesis

In view of what has already been said about the general pathogenesis of peritonitis it is necessary in this place to call attention to those points which have specific application to the appendix only. We are here less concerned about the primary source of the infective process in the appendix, since that has been discussed in the section on etiology, than about the process by which the inflammation reaches the surface of this organ, once it has begun. The chief discussion centers about the processes involved in the extension of the infection to the surrounding peritoneum. In every appendiceal inflammation recognizable clinically, this organ's own peritoneal covering is involved in a reactive process by direct extension, otherwise the condition could not be diagnosticated.

The problem involves the establishment of general laws from a series of observations. From the determination of the stage of

the disease from the clinical history we obtain an idea of how long a time was required for a certain anatomic state to be reached. By comparing a large series a fair knowledge of the pathology as a sequential process is arrived at.

In all cases there is more or less involvement of the various coats of the organ which finds expression in round-celled infiltration, edema, and fibrinous exudation. This fact gives little evidence of the nature and extent of the primary infective process. All coats of the entire appendix may be so affected, even in cases where the bacteria are confined to small foci between the lymphoid nodules in the submucosa. In such cases the peritonitis is strictly speaking a bacteria free inflammation, and in a clinical sense an aseptic peritonitis. That is to say, the exudate within and upon the peritoneum is free from bacteria. Our concern here is to determine the nature of the changes which must take place in the organ before the infecting organisms can reach the surface of the organ and become a menace to the general peritoneal cavity. The appendix is such a serious menace, it may be insisted on at the outset, not because it is an organ made up so largely of lymph tissue prone to infection, but because it has a precarious blood supply. Being dependent on a single terminal vessel, when this is affected by thrombosis, destruction of the walls of the organ must follow.

As has already been discussed in the general section of pathogenesis, the degree of change in the wall of a gut necessary to admit the passage of bacteria can not be definitely stated. Confusion has arisen because clinicians have assumed the escape of bacteria when an aseptic floccular exudate was formed about the appendix, or if an exudation existed in surrounding organs. We have been led to assume the escape of bacteria when no escape has taken place. That bacteria escape through the walls of the appendix when there is no macroscopic lesion there can be no doubt, but that they do escape as readily as generally assumed remains yet to be proved.

Sprengel noted such an extension in simple appendicitis in 3 of 15 cases, and Krogius saw such extension in 6 out of 46 cases. Some observers believe that such extension is possible even when the wall of the appendix remains intact. An entirely intact wall

is evidently not meant, since reference is made to a septic lymphangitis, to which is ascribed the capacity of carrying organisms to the peritoneal surface. So analyzed there is obvious agreement that, in order that infective material shall escape from the appendix to the surrounding tissues, the wall must be organically or functionally changed, for unless there is some physicochemical alteration in the component structures of its wall, bacteria can not pass. I have sought to compare the histochemical structure of these appendices, which have permitted the escape of bacteria, with those, obviously inflamed, which have not permitted bacteria to pass. Obviously, in pursuing such an investigation we have available for study only the state of the organ at the time of observation. From a study of a large series of these we are enabled to form some idea as to the extent of the process which must precede the escape of bacteria.

In general it may be said that so long as one or more coats remain unchanged, as indicated by their acceptance of specific dyes, when the cellular exudates in the interstices show no nuclear degeneration, and the fibrinous exudate is fibrillar, bacteria do not pass. When the fibrillæ no longer take acid stains, and when the exudate consists of a granular fibrin with associated necrobiotic leucocytes, bacteria may pass. They are likely to pass when there is an adjacent fibrinous exudate in surrounding organs; that is to say, an appendix of the structure just noted surrounded by an infiltrated omentum encourages the escape of serum toward this omentum, and with this escape of serum bacteria follow. Whenever there is an adhesion, therefore, bacteria are more apt to escape than when there is no adhesion, though of course escape under such conditions is less deleterious to the patient. This interchange between diseased organ and a surrounding adhesion seems to have the twofold purpose of admitting defensive forces, leucocytes and serum, to the suffering member and of encouraging the escape of bacteria to the succoring organ where, advancing in open ranks, they can be more effectually dealt with. The effect of the omentum on a diseased area may be imitated with a pledget of gauze which, when it becomes infiltrated with leucocytes, has many of the functions of the normal omentum. This admits of the study of the bacterial flora at any stage of tissue change in the diseased

area. The effect of fibrinous exudate on the migration of infection is a matter of daily observation. Surgeons employ this law in the drainage of abscesses. They know that if an abscess is not actually opened into by the incision in the course of time the abscess may find the drain opening. This happens because the drain tract made by the surgeon sets up an aseptic reactive process which sets up an interchange of serum between this opening and the abscess. Bacteria making use of this avenue of communication extend the abscess in this direction and escape of pus through the previously dry opening follows. It may be remarked that this is more apt to occur if a gauze drain is used than if a rubber one has been used, because the gauze produces a greater leucocytic and serous infiltration about it.

### Classification

It is problematic whether an attempt at classification is worth while in a disease showing such indefinite dividing lines as the various "types" of appendicitis. As a concession to the clinician such a classification will be adopted, though it must be insisted that appendicitis, like the road to perdition, consists of a series of changes which unchecked, by natural or artificial aid, leads finally to destruction. Many classifications for appendicitis have been proposed, some from the point of view of the pathologist, and some from that of the clinician. Lennander applied the simple term *acute* to all forms, provided the case under consideration is not subacute or chronic. This classification does very well as a pre-operative diagnostic classification, but once the abdomen is opened the surgeon must apply a much more discriminating analysis. Sprengel divides them into the simple and destructive types. This classification has been commended by a number of surgeons and is popular among physicians who sometimes advise operation and sometimes do not. Giertz augments Lennander by the addition of the amplifying term *perforative* or *gangrenous*, as the case may demand. Kelly divides them into catarrhal, diffuse, purulent, gangrenous, and perforative. This classification from the pathologist's point of view is excellent (if there is such a thing as a catarrhal type), but, in so far as it relates to the general peritoneal cavity, it is too prolix. Since the chief interest in the appendix cen-



ters on the relation of its disease to the general peritoneal cavity, that factor should be made the basis of classification. A simple classification of the material which comes to the surgeon's hands may be suggested as follows: the diffuse exudative, the ulcerative, and the gangrenous. Whether these classes are final stages, or whether the one merges into the other, depends on the aid received from the defensive forces and the complications arising within the appendix itself.

**The Diffuse Exudative.**—Beginning at one or at several points in the wall of the appendix, usually in depressions between the lymph follicles, bacteria lodge and leucocytes collect about them. It is interesting to note that, in many instances at least, while the entire lymphatic apparatus of the appendix is in a state of reaction there may be but a single focus actually harboring bacteria, though of course the lumen of the appendix harbors a great variety of bacteria, as it normally does. In response to this local infected focus there is a general hyperemia of the entire organ, both the service and the potential vessels being dilated markedly. If the process does not go beyond this stage, regression may be prompt and the peritoneal involvement will not exceed an active hyperemia (Fig. 176). If the process is more intense a general edema of the organ results (Fig. 177) with the production of pain, due to the stretching of the nerve plexus. In this state the surface of surrounding organs, particularly the omentum, may respond with hyperemia and exudation. The exudate may be sterile or it may be infected with nonpathogenic bacteria or with such bacteria as possess but little virulence. Regression may take place at this stage within a few days. This constitutes the so-called catarrhal appendicitis, or appendicular colic, leaving but little evidence of the disease. This type might better be called the intermittent or remittant form. After an inconvenience or disability of some hours or days the patient proceeds about his business.

When the primary focus extends, all of the walls of the gut may become involved and the surface of the peritoneum may be reached by the abscess. The omentum usually takes part (Fig. 178) as above noted and in a week or ten days a periappendiceal abscess is formed (Fig. 179). Preceding this there is an intense change in the tinctorial reaction of the tissues of the wall of the appendix,



Fig. 176.—Early acute appendicitis with edema of the walls. There is beginning distention of the vessels of the appendix and cecum. This was the second attack. There are abundant adhesions from a previous attack.



Fig. 177.—Early acute appendicitis. There is edema of the walls, hyperemia of the peritoneal layer and some exudate. The patient had had a number of mild attacks. The organ was hard to the touch and was erectile.

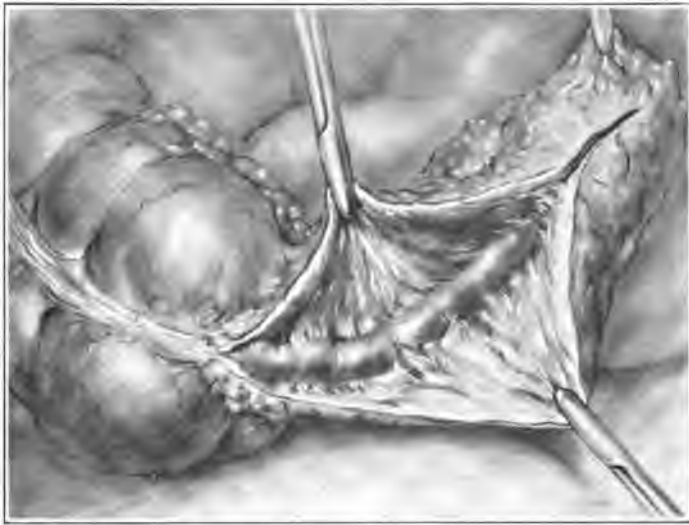


Fig. 178.—An acutely inflamed appendix entirely surrounded by the indurated omentum. The proximal portion of the omentum was severed allowing it to retract before the sketch was made.



Fig. 179.—Appendix in which the wall is much increased in thickness due to several small abscesses.

possibly with occlusion of the lymph stream by coagulated lymph or dead leucocytes (Fig. 180). Blood vessels may become occluded in a like manner. Bacteria may now escape without there being

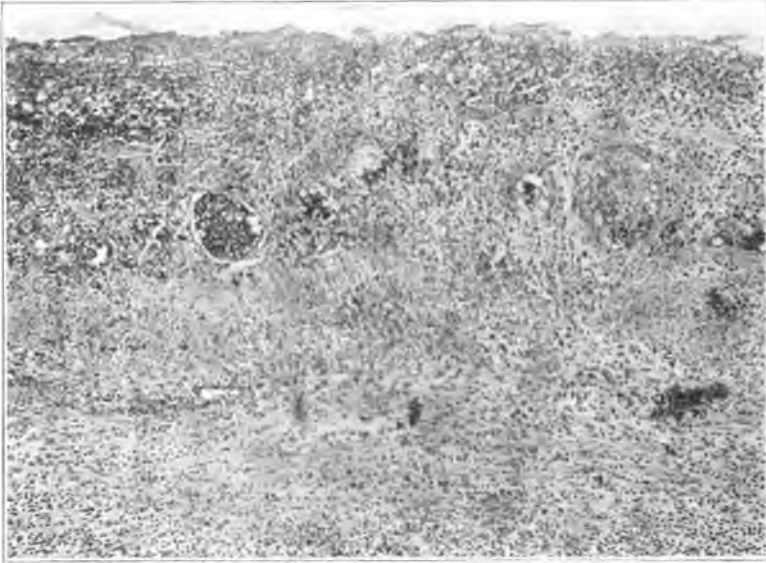


Fig. 180.—Acute appendicitis showing lymph and blood vessels filled with clots.



Fig. 181.—Acute appendicitis in which a small gangrenous area about to perforate is seen near the blackened and thickened extremity of the organ.

a macroscopic opening or there may result a local necrosis of the appendix wall with an escape of the bacteria-laden contents of the appendix upon the free peritoneal surface. A periappendiceal

abscess is the result provided adhesions with surrounding organs have already developed. This process may take place so rapidly that the walling-in process has not yet taken place and a diffuse peritonitis may result. In such instances a gangrenous area visible to the naked eye is usually present (Fig. 181). In this condition there is usually a thrombosis of one of the larger branches of the appendiceal artery. In such cases there is no sharp distinction between this type and the frank gangrenous variety except in extent of involvement. This process can be traced only by studying the entire organ carefully. At a point remote from the chief focus of destruction its supplying vessel will be found thrombotic. As a result, while the peritoneum of the appendix at some distance from the necrotic area may be excited to plastic exudation with adhesion to the environment, that portion of the organ proceeding to gangrenous change repels any familiarity on the part of surrounding organs. These are the cases that apparently do fairly well for a number of days when they suddenly "go bad." In such cases after the finger of the surgeon has separated omental adhesions from the base of the appendix, its more degenerated extremity pops into the field of vision.

**The Ulcerative.**—Those cases in which there is a local ulceration with relative freedom of the remainder of the organ may be considered under this head. It may take place in the presence of a foreign body or without such an exciting factor. In the latter instance the wall surrounding the ulcerous area may resemble an acute perforative peptic ulcer. The entire thickness of the wall is destroyed because of deprivation of nutrition or by the destructive infiltration of leucocytes (Fig. 182). It is as though a boil formed within the wall of the appendix subsequently breaking through the peritoneal surface. To all these instances of course the whole appendix is somewhat changed but not enough to excite protective adhesions. The result of a solution of continuity of the entire thickness of the wall is the escape of the entire flora of the appendiceal lumen. The surrounding peritoneum is not prepared for the infection, adhesions have not taken place, and the exudate has free passage among the intestinal coils or wherever peristalsis, capillary attraction, or gravity may lead it. This process takes place most frequently at the base of the appendix or near its tip.

In this type there is an initial mass destruction of the entire wall, producing, so to speak, a punched-out opening. The evidence of this is to be found in the walls of such ulcers. Necrosis of tissue without the presence of large numbers of leucocytes is the characteristic feature. This may be brought about either by occlusion of vessels supplying this area or by the presence of bacteria which have the power of liquefying tissues and at the same time acting

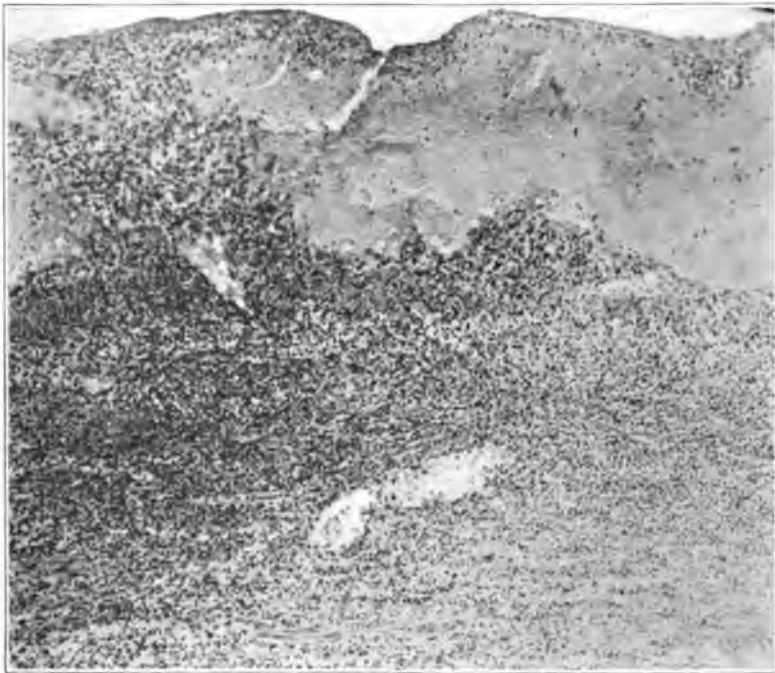


Fig. 182.—Section from the wall of an appendix near a perforating ulcer.

as repellent to the protective agents. The significant factor in this type is that there results an open passage between the lumen of the gut to the peritoneal cavity without the intervention of protective adhesions. Even when the perforation is abrupt there may be a secondary reaction of the surrounding peritoneum however which may obscure the pathologic picture, but it is to be remembered that bacteria and other contents of the appendix have escaped and drainage is required.

When a foreign body exists there usually is a necrosis coextensive with the foreign body. In such cases the first evidence of a peritoneal involvement may be an escape of the foreign body into the peritoneal cavity. This perforation may take place so suddenly that the wall is so little changed that it actually rolls as if incised. It is interesting to note how tensely the muscle coats grasp such foreign bodies. When incised in the little inflamed organ the foreign body may actually pop out (Fig. 183). Slight infection must lead readily to perforation. Often however infiltration precedes the final perforation, and not infrequently complete walling off takes place so that the foreign body is found in the abscess.



Fig. 183.—Large enterolith in an appendix. The appendix was removed a few hours after the initial pain. When cut into, the muscle walls retracted forcing out the foreign body.

**The Gangrenous.**—In this type there is death of all or a large part of the appendix occurring simultaneously. The entire organ is black and shiny resembling the colored gentleman as he makes down the Pullman bunks on a July evening, and is generally but moderately thickened. This type is the product of the occlusion of one of the chief supplying vessels of the appendix (Fig. 184). In structure such an appendix finds an exact counterpart in the intestine in mesenteric thrombosis in the walls of an ovarian cyst with a twisted pedicle. In such cases the tinctorial reaction of the gut walls may be astonishingly little altered, and there may be slight

cellular infiltration in the walls of the organ, being confined usually to rows of leucocytes arranged between the fibrils of the submucosa or subserosa. The fibers usually suffer no tinctorial changes. The extent of cellular exudation depends upon the suddenness and extent of the thrombotic process in the supplying vessels. There is in the beginning, at least, nowhere a solution of continuity of

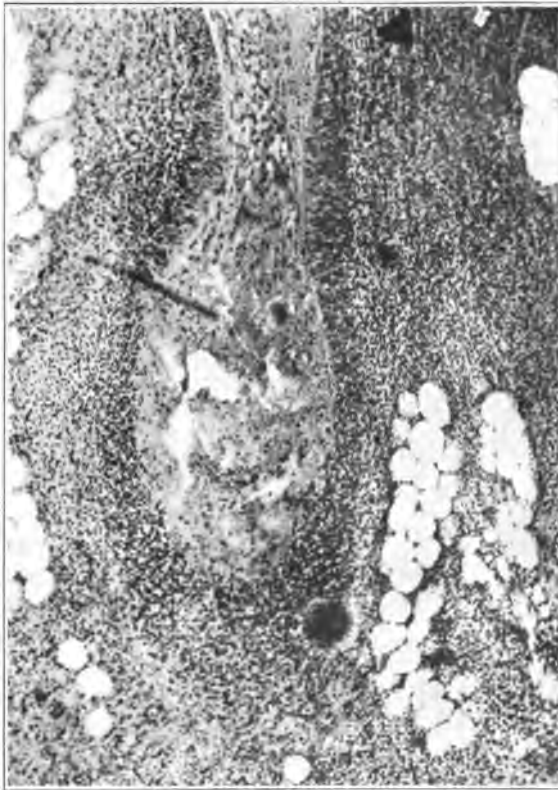


Fig. 184.—Thrombosis of the mesenteric artery in a gangrenous appendix. There is marked perivascular infiltration.

the wall and hence no escape of its contents. When the appendicular artery becomes occluded the organ may be but little increased in size and may feel soft, even semifluid to the touch. The necrotic organ, coming into contact with the surface of surrounding guts, excites them to exudation of a nonplastic fluid. Early in



the course of the disease this exudate may be sterile and so great in amount that the blanched organ fairly floats in it. The subsequent course of this type can not be predicted with certainty. Secondary destruction of the walls may take place permitting of the mass escape of the contents of the appendix. This state is reached after the necrotic area is separated from the living part due to processes going on in the part of the organ not involved in the necrotic process. Usually some days are required for this stage to be reached. A considerable part of the organ may become absorbed, leaving little but the meson to indicate the site of the organ. Whether or not such an organ once necrotic is capable of reestablishing the circulation is difficult to say. I have examined several such specimens which suggested that reestablishment of the circulation was in progress. This seems possible. If the omentum or other organs are adherent to it they may be able to supply nutrition during the period of stress, just as takes place when the pedicle of an ovarian cyst becomes twisted. It attaches to the surrounding peritoneum until its pedicle functions again when the adhesions are released. However, because of the nature of the changes in the walls of the appendix adhesions do not readily take place.

The significance of this type is that, notwithstanding the striking appearance of the appendix and the presence of the exudate, in the early stage no intestinal contents or infection may have escaped, and the affection may really be an innocent one. The period that must elapse before perforation takes place is considerably greater than in the ulcerative types, and is comparable with mesenteric thrombosis and mesenteric strangulation in hernia.

### Pathology

In discussing the pathology of the appendix some of the points discussed in pathogenesis must be repeated. The pathology is made up merely of a series of observations from which the attempt was made to chart the disease as a process.

It seemed expedient to divide the diseases of the appendix into three categories when discussing the pathogeneses. The division is artificial, but it seems to typify certain general end-results as the surgeon sees them in the operating room. In the laboratory it at once becomes apparent that such a division is in a measure ar-

tificial, because all inflammatory diseases of the appendix are identical in a general way in their pathology, and the clinical end-results depend upon what are sometimes very slight variations in detail. Similar wide variations are observed in the diseases of the tonsil; follicular tonsillitis, peritonsillar abscess, malignant endocarditis, or polyarthrititis, so wide in their significance, are all the products of similar changes in the tonsil.

The first thing that impresses one in the study of the pathology of the appendix are the slight changes that remain after very marked acute inflammation. I have repeatedly observed instances where an appendiceal abscess had been drained and extensive induration noted, the appendix when removed after some months showed very slight changes when viewed under the microscope. A few lingering plasma cells, or an increase in fibrous tissue may be all that is observed. On the other hand, the most pronounced evidence of necrosis may show but little under the microscope. It seems best to discuss here the findings in certain types of appendices with but little speculation as to how they arrived at the state they present.

**Atrophy of the Appendix.**—In some instances the various layers of the appendix retain their normal relations but each is much reduced in volume. These are usually found in individuals with fat mesenteries. The appendix lies in a groove in the fat meson. This fat is usually very dense to the touch, without any evidence of inflammation. I believe the general atrophy is due to the gradual narrowing of the lumen of the vessels from the ever increasing pressure of the fat upon them (Fig. 185).

**Fibrosis of the Appendix.**—The external appearance of this form may resemble the atrophic. In this type the typographic relation of the various coats is lost, due to the partial or complete destruction of one or more of them. Usually the mucous coat has been lost and a fibrous tissue has displaced it. In this way the lumen becomes obliterated by fibrous tissue, resembling scar tissue in the caliber of the fibers and the sparseness of the nuclei. Sometimes but a part of the lumen is so obliterated and there results a dilatation of the distal end, leading sometimes to the formation of large mucous cysts. The muscular coats in this type may be much re-

duced in thickness and replacement by fibrous tissue may be in evidence.

In some instances the evidence of destruction may be much greater. A part or all of the appendix may be represented by a thin fibrous band lying along the edge of the meson. Usually there is a segment of the appendix at the cecum and often the terminal end remains. This remaining end may feel like a lymph gland lying at the tip of the meson. These portions remaining may have the appearance of a normal appendix.

**Catarrhal Appendicitis.**—This term implies that there is a dis-

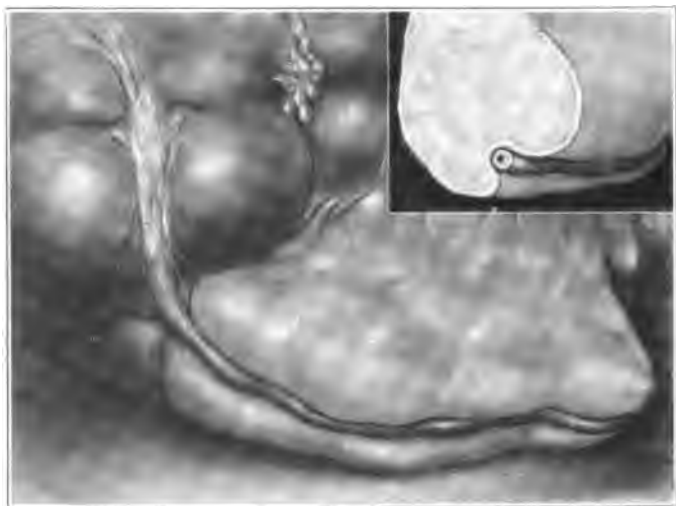


Fig. 185.—Atrophic appendix imbedded in a fatty mesoappendix.

ease of the appendix characterized by an increase in the function of the mucous glands. It is possible that the appendix shares in the activity of a mucous colitis. There is no evidence that there is an isolated catarrhal lesion of the appendix. This term was hypothesized to include those cases characterized by brief pain and soreness in the region of the appendix. Specimens secured at this time show that there is really a deep-seated inflammation present and there is no evidence of a catarrhal state.

**Diffuse Exudative.**—The focal lesion in the appendix, as in the tonsil, is an area of infection between the follicles. This is sit-

uated just below the surface mucous layer and involves it secondarily.

It is a question whether infection reaches this site by extension from the surface of the mucosa or through the blood stream. Both theories have been championed.

In some instances no doubt bacteria reach this site by direct extension from the surface. The extension can be directly traced when foreign bodies lie at the point of infection. There is increasing evidence on the other hand that at least in many instances the infection reaches the appendix through the blood stream. Not infrequently appendicitis follows close on an attack of tonsillitis. In such instances there is a distention of the lymph vessels, probably dependent on a general thrombotic process, since many follicles are similarly and equally affected, which would likely not be so if the condition were dependent on a local source of infection. In the majority of cases there is no such association with a primary focus situated elsewhere in the body.

It is difficult to determine the number of primary foci because a search of the entire organ involves immense labor. Aschoff is of the opinion that there are many primary foci in all cases. I do not believe that this is true. If those organs are examined in which but a portion of the organ is diseased, one can find the primary focus at the proximal end of the diseased area. In determining the point of origin one must distinguish between simple leucocytic infiltration and the primary bacterial focus. The leucocytic infiltration and exudation of serum involve, more or less, the whole organ, while there may be an infection at one point only, just as a felon causes swelling of the entire thumb while the actual infection involves only the periosteum of the terminal phalanx.

In early cases, areas of leucocytic increase about the lymph follicles is all the change noted, aside from the general edema which gives rise to the symptoms which made the diagnosis of the lesion possible. In mild cases the disease may regress at this point, giving rise to the mild cases referred to as catarrhal. These cases find their counterpart in follicular tonsillitis. When the process lasts longer there is a further increase in the leucocytes about the area of infection and its center may show some degeneration, while the muscular coats show an infiltration of leucocytes and there is

an exudate on the surface of the appendix. These changes occur when there is local tenderness and muscular rigidity. In more advanced cases there are adhesions about the appendix with an increase of all the changes above noted. There is often hemorrhage into the walls of the appendix, particularly into the subserosa. The mucosa is often exfoliated and there may be a distinct breaking down in the foci primarily involved. This may be considered the height of the process in cases running their course in ten days to three weeks. When these cases have run their course all that remains to indicate past trouble is some plasma cell infiltration, possibly some increase in the germinal centers, and here and there some scarring in the submucosa.

In the type where a periappendiceal abscess forms there is an extensive exudation of granular fibrin, degenerated leucocytes, and finally molecular disintegration of the muscular and serous coats. In these cases there remains after recovery a scar extending through all the coats of the organ, and there is often evidence of chronic vascular changes.

When the focus is more virulent, or thrombosis in a vessel occurs, involvement of the entire thickness of the appendiceal wall takes place. This area is wedge-shaped with the base of the wedge away from the meson. The reason for this shape is that all gut arteries are essentially end arteries, just as one sees them in the kidney and spleen. The primary change is an anemic necrosis. The area so involved becomes separated from the surrounding viable tissue, and a perforation, "punched out" in appearance, occurs. This state can be observed by sectioning these openings, keeping in mind the appearance of sections of like processes in the spleen and kidney. When this occurs, the gut contents escape into the surrounding peritoneum, if protective adhesions have not formed before this disaster occurs.

Perforation may occur from the rupture of abscesses situated in the wall of the appendix, in the absence of thrombotic processes. In such cases the perforation is not so precipitous, and protective adhesions usually occur with the result that a localized abscess is formed.

Bacteria may escape in such conditions without actual perforation. Fibrin is deposited on the surface of the organ and the en-

tire wall becomes infiltrated with fibrin, and osmotic processes toward the periphery are set up and bacteria are conducted beyond the appendix. In this type bacteria are distributed over a wider area than in any other type.

**Gangrenous.**—The third type, the gangrenous, is dependent on the occlusion of the chief supplying vessel. The changes here are analogous to those which occur in the walls of an ovarian cyst when the pedicle becomes twisted. The infection here may be limited to the proximal region of the appendix, in which event the entire appendix is involved, or may occur more distally, in which event only a part of the organ is black. The blackness is due to extravasation of blood and not to degeneration of the tissue. Such appendices take all dyes clearly early in the disease, which indicates that lytic processes are not active. Such organs, like the cysts with twisted pedicles, accept temporary aid from all organs which come in contact with them. In such cases the organ shows no changes on microscopic examination except the extravasation of blood.

In these cases in which there is complete exclusion of nutrition there is a disintegration of tissue, the cells no longer take any stain, and the fibrous tissue loses its specificity or may take a basic dye. In such cases there is no restoration, and if there are no adhesions the lumen of the appendix comes to communicate with the peritoneal cavity after the necrosed tissue becomes separated off.

The surface of a black appendix may be free from bacteria, until a perforation occurs. After the first acute disturbance these organs may hang free in the abdomen with but little disturbance attending them.

### Symptoms

The so-called cardinal symptoms of appendicitis are familiar to every one. They consist of pain, vomiting, local tenderness, and fever. The general characters of these phenomena belong to all types of acute peritonitis, and they have already been considered, but their special characteristics as observed in appendicitis remain to be considered.

**Pain.**—The pain is generally sudden in its onset, often emphatically so, warranting the appellation “*coup de Pistolet*,” a term

applied by several French writers. This is particularly true when there is sudden perforation or complete occlusion of the artery with subsequent gangrenization of the entire appendix. In cases in which perforation is sudden, the pain may be so acute as to cause collapse, or, at least, collapse is associated though possibly it is caused by the escape of gut contents. On the other hand the beginning may be marked by but little pain. This is particularly true when there is recrudescence of a slumbering lesion.

In the earlier stages the pain radiates over the abdomen, and is very liable to be most intense in the region of the umbilicus or in the epigastrium. This pain is reflex in character, and may be caused in some instances by irritation of the mucosa with the associated contraction of the muscle. In the majority of instances it is caused by stretching of the nerve plexus from the developing edema. Whether this explanation is correct or not the fact remains that if the appendix is cut into during appendectomy under local anesthesia and a forceps put into the lumen of the appendix, and the blades then separated, the patient experiences pain in the epigastrium, and may be made to vomit. There is certainly no longer any excuse for assuming that the diffused pains are caused by an early diffused irritation of the peritoneum which later becomes localized in the region of the appendix. Operations during the period of the diffused pain have failed to disclose any diffuse irritation. Possibly spasmodic contraction of its walls in an effort to expel some of the contents may be an added factor in such instances. That bowel contents may enter the lumen of the appendix is abundantly proved by the bismuth test meal. Whether painful contractions can be excited by these contents is less susceptible of demonstration. The bismuth test meal indicates that the normal appendix rids itself of such foreign material without attracting the attention of the higher centers.

These preliminary pains are intermittent in character with periods of more or less complete remission. At their height, or even in their incipency, they may be attended by nausea and vomiting. These initial pains are not referred to other parts of the body, and they give no clue to the location of the organ which excites them.

Sooner or later, usually in from six to twenty-four hours, the generalized pains lessen or cease, and the right iliac fossa becomes

the site of the greatest distress. Often before spontaneous pain in the region of the appendix is complained of, pain may be elicited by pressure in this region even before muscular rigidity is apparent.

The distinctive pain of appendicitis is due to the irritation of its surface and of the surfaces with which it comes into contact. When there is no escape of contents of the appendix the entire disease process tends to remain so localized. When its contents escape into the general peritoneal cavity the distinctive features of periappendicitis are lost, and the whole picture merges into that of a diffuse generalized peritonitis. Even then, certain features may stand out distinctive of the source of origin, either of diagnostic or therapeutic importance. Among these may be mentioned a greater degree of tenderness, rigidity, or edema in the right lower quadrant. Usually, however, the history is the most reliable guide to the origin of the infection.

With the advent of periappendicitis the spontaneous pain becomes localized at the site of the lesion. Since the organ usually occupies the iliac fossa the pain is usually here. When the organ is located in the pelvis and its tip is chiefly involved, vesical or rectal tenesmus, particularly in children, may be the dominating symptom.

When the appendix is turned upward laterally to the colon, the pain may be in the region of the gall bladder. When the appendix expatiates itself into a hernial sac and becomes diseased it expresses its complaint in the language of its adopted land. I saw one patient, a boy of four, in whom the pain was felt in a left inguinal hernia, the sac of which the inflamed appendix occupied. There is on record a case of appendicitis occurring in the left pleural cavity, the organ having gained access to this cavity via a diaphragmatic hernia.

When the appendix lies retroperitoneally the peritonitic pains are absent, and the picture becomes that of a retroperitoneal infection. Usually a deep tenderness and edema are all that mark the site of infection. The various types of appendicitis cause a variable amount of pain. The large edematous appendices of the nonperforative type cause the greatest degree of irritation to the surrounding peritoneum and, in consequence, the greatest pain. This



is apt to continue for a considerable time because the surrounding tissue partakes of the tendency to serous infiltration.

With this type the peritoneal irritation is associated with a serous exudate in the general peritoneal cavity, as well as in the subserous tissue. This fluid may be considered in the nature of a by-product. It rarely becomes so great as to be readily demonstrated clinically, neither does it exert any influence on the course of the disease. If clear in character, it indicates the absence of diffuse infection, though it may be so loaded with leucocytes and fibrin flakes as to impart a cloudy appearance to it, and yet may be non-infective in character. The type of cells is similar to the type of those within the walls of the appendix, and, obviously, represents the amount of fluid which found no space within the meshes of the tissue.

The amount of fluid is generally inversely proportional to the amount of pain. In some instances, particularly in children, the fluid may be so abundant as to be of diagnostic significance. This is especially true in young children who do not reliably indicate the site of greatest pain and in whom general abdominal distention tends to obscure muscle rigidity. By rolling these little patients from side to side dullness may be demonstrated in the flanks, in this way revealing the presence of this type of appendix.

When there is perforation without previous periappendiceal adhesions the contents of the appendix escape. When abundant, a direct foreign body irritation may be added to that of the infection. In such cases the pain may rival in intensity that of a perforating duodenal ulcer, and like a perforating ulcer, may be intermittent. In these cases a huge amount of semipurulent exudate may form in a very short time.

The pain in the gangrenous type is characteristic. The initial pain is very intense and is continuous, but after persisting for twelve hours or more, it subsides. The explanation seems to be that as soon as the vessel is shut off the tissue dies, and the pain is that of acute necrosis, and as soon as the nerves die pain ceases. When perforation follows necrosis, renewed pain, that of peritonitis, supervenes and then continues as a primary perforative peritonitis.

Those cases which begin with intense initial pain are in general

of serious character, but not all of the serious cases are attended by severe pain. Pain is a measure of irritation, while danger to life is dependent on the toxicity. When intensely septic material escapes from the appendix there may be little pain because the character of the infection is such as to repel all reactive processes and therefore may be quite pain-free. As a matter of fact the moribund patient is usually free from pain and his distress comes only from distention, vomiting, and like secondary phenomena.

Spontaneous pain is one of the most reliable symptoms in the diagnosis of the disease, and while the severity of the initial pain may give a general clue to the severity of the attack, once the initial pain subsides, the sensations of the patient are wholly unreliable as an index to the course of the disease. When pain is relied on as a guide to the time for operation disastrous errors will be committed.

**Pain on Movement.**—Coexistent with the spontaneous pain is pain engendered by movement imparted to the affected area. This may be produced by movements of the body as a whole or by parts of it. Jarring of the body, as in the movements imparted to the bed or to the conveyance upon which the patient is being transported, may excite it. This is caused by the change to its environment imparted to the inflamed organ by the sudden motion of the body as a whole.

The most common source of increasing pain is movement of some part of the body against the inflamed mass. This may be the movements of the intestines transmitted from the diaphragm in respiration, the contraction of the psoas in walking, or of the bladder or rectum as they dilate and contract in the performance of their functions.

The location of the pain so elicited gives a very accurate clue to the location of the diseased organs. The most certain evidence from these signs is obtained when they are produced by the patient's own volition. The stooped gait with the body inclined slightly to the right or the drawn-up right thigh as the patient lies in bed declares eloquently that the site of the lesion is over the psoas muscle. The precautions that the patient takes to limit motion of the affected area may not be particularly noticeable in the respiratory movements. When the respiratory movement is en-

tirely costal it is at once apparent, but, on the other hand, the diaphragmatic movements may be carried out with caution so that its excursions are regular and calculated, but limited. The right half of the diaphragm, and with it the abdominal wall, may make shorter excursions than the left. This phenomenon is best discovered by allowing the light to fall over the shoulder of the observer, as he stands at the foot of the bed, upon the bared abdomen of the patient. When not apparent at once it may become so if the patient is encouraged to breathe more deeply. This sign is of value in children, particularly before the distention of the abdomen has become very great.

After the disease has existed long enough for the adjacent parts to become fixed to each other, the movements above noted do not cause so much pain because movement of the entire mass results.

The presence of pain on movements imparted to the affected area is quite as important in revealing complications as in the primary disease. Subhepatic pain or subdiaphragmatic pain developing in the course of the disease speaks for extension in that direction, as does vesical or rectal irritation, not previously present, for extension into the pelvis.

**Pain on Pressure.**—Frequently as the surgeon approaches the patient for the purpose of palpating the abdomen the patient expresses his belief in the presence of local tenderness by involuntarily extending his hands in protection. Being possessed of this advance knowledge it is often well to allow him to complete the examination and indicate to the surgeon the point of greatest tenderness. A systematic perusal of this plan will soon convince the observer that the initial point of greatest tenderness varies greatly from McBurney's point. Like most great men who generalize a great truth, McBurney himself stated that the point of maximum tenderness may vary from this point but, many of his followers, to simplify the conception, have taught that the sensitive point must be there, and they have entered into prolonged ptolematics to prove that it is so. For this reason I believe Cordier's characterization of the designation of this point as the location of maximum pain as "most unfortunate" is not far from the truth. However, the observation of McBurney called attention to a great truth, and was a great factor in teaching the profession the early diagnosis

of periappendiceal peritonitis. Having learned the fundamentals from a dogmatic statement it is proper to proceed to a refinement by recognizing the fact that the initial pain in appendicitis may be at a considerable distance from this point. The great variability in the location of the appendix was noted in the chapter on anatomy. The portion of the appendix affected likewise may influence the point of greatest pain. The tip of a long appendix may lie beside the rectum in the culdesac and give rise to vesical and rectal tenseness while the usual site of appendicular pain is free from disturbance.

It is well to remember the significance of a more or less localized tenderness, usually somewhere in the right lower quadrant, but to overlook the possibility of appendicitis when the initial pain is elsewhere is to court frequent error. It is instructive to the surgeon to note the exact site of local tenderness on his clinical examination and then note the exact anatomic location during the operation.

Much discussion has arisen as to why the maximum pain is not always located at the point of greatest pathologic change. The reason is that it is the degree of irritation produced that governs the amount of pain and not the extent of pathologic degeneration. An omental mass adherent to a gut, or particularly to the abdominal wall, will very likely determine the point of greatest tenderness while the site of greatest pathologic change in the appendix may lie at some distance. When a diseased appendix which has excited the pain is examined histologically one can not wonder that the location of the appendix does not always correspond to the site of maximum pain for it may be wholly degenerated. Poets tell us grief is sometimes too great for tears, and an appendix may be too rotten to hurt, Morris, as usual, drives the tack with a sledge hammer in these words, "The reason why the appendix is free from tenderness is, because it is dead, nerves and all." There is no need to invoke special sensitiveness in certain regions. If the site of greatest pain is determined with the aid of the patient, and then at operation all the pathologic changes are carefully noted, the location of abdominal pain as relates to intraabdominal changes will become much simplified and there will be no need to invoke a complicated reflex process to explain its location.

The statement of Morris above quoted gives the clue to the correct understanding of those cases, which, in spite of extensive changes, are painless both spontaneously and on pressure. In very virulent infections the organ becomes necrotic without preliminary reaction. In the gangrenous type there is intense initial pain—then all is quiet. The reason is that the whole organ is totally dead, and being dead it excites no painful impulses. Appendices, like dogs, when once thoroughly dead, do not bark. Appendices, like canines, when dead for a certain length of time, may become offensive again through the very fact that they are dead. The degenerating appendix may in this state not irritate the surrounding peritoneum until disintegration permits the escape of its contents. I once saw a young man who was stricken with sudden excruciating pain low in the groin which diffused over the abdomen. In twenty-four hours the pain had subsided. On the fourth day a new pain gradually developed well above McBurney's point. At operation a long black appendix was discovered. The cecum at the base of the appendix had begun to separate, and bowel contents were beginning to ooze out, exciting the surrounding peritoneum to reaction.

Superficial tenderness, emphasized by Dieulafoy, is an uncertain sign. Early in the disease it may be caused by a reflex through the sympathetic. It is a very unreliable sign because it is present in so many neuroses. This type of pain will be discussed under chronic appendicitis.

**Muscular Rigidity.**—This sign is but a corollary to the preceding. The careful respiration and flexed thigh are associated with rigidity of the rectus muscles. As already indicated, the lessened excursion of the abdominal muscles in respiration may be determined by the naked eye. The palpating finger of the surgeon emphasizes the impression gained by sight and detects the lesser degrees not apparent to the eye.

The rigidity of the muscle is a reflex designed to protect the diseased area from pressure and from movement. The same phenomenon is noted in the free hand of a person affected with a felon on his thumb when a solicitous friend inquires as to the cause for the draping of the digit. Speculation as to the character of the nervous mechanism involved is not profitable.

The rigidity is greatest over the area of maximum involvement of the parietal peritoneum. This usually includes the lower segments of the recti and the lateral abdominal muscles. When the appendix lies far lateralward the posterior group may show great rigidity. When located high up under the liver the upper end of the rectus alone may be rigid. When the inflamed organs become conglutinated, with or without attachment to the abdominal wall, so that movements of the abdominal muscles will not cause an increase of pain, the rigidity relaxes. The relaxation begins in the regions most remote from the site of maximum irritation. Because of this a mass produced by the inflammation becomes easily palpable when the rigidity ceases.

It is interesting to note that as general muscular relaxation occurs as the patient goes under an anesthetic, the parts of the muscles which lie directly over the lesion relax last. Because of this fact the surgeon, laying his hands gently over the abdomen of the patient as he goes to sleep, may accurately judge the site of the greatest intraabdominal irritation, which in the early cases usually means the site of the appendix.

Rigidity may be absent. If the condition of the appendix is such that no irritation is imparted to the surrounding structures, the muscles do not respond because they get their clue only from painful impulses.

When the patient is in extremis, especially when due to cerebral irritation, the abdomen may be retracted and the muscles seem rigid. This rigidity is uniform and does not characterize any particular kind or location of lesion.

**Vomiting.**—Patients with appendicitis often suffer from disturbances of the stomach, varying from an uncomfortable, burning sensation to violent and protracted vomiting. Vomiting is a common symptom, but loses in importance because it is an accompaniment of so many other conditions. It is only when it is associated with localized tenderness and muscular rigidity that it becomes of value in diagnosis. Save in the violent hemorrhagic types, vomiting gives little evidence as to the severity of the disease.

The cause of the vomiting is generally ascribed to inflammatory irritation of the peritoneum. This view is held despite the fact that vomiting usually occurs before such irritation begins and

ceases before the irritation is at its height. It seems to me clear that the phenomenon is reflex. I have already stated that vomiting may be produced by artificially distending the appendix. Simple traction on the appendix, as in attempting to pull it into a wound made for a gall-bladder operation, will uniformly produce nausea and even vomiting if persisted in. The same is true of traction on the gall bladder, colon, or jejunum, as I have repeatedly observed in doing gastroenterostomies under local anesthesia.

The amount and character of the vomiting varies. Usually a little mucus or food is expelled. Bile is rarely produced, even when retching continues for some time.

In rare instances blood is vomited. Sprengel saw it in no less than nine cases. The cause of this condition has been discussed here in the section on general symptomatology and in that on complications after operation.

Recurrent vomiting sometimes takes place. This may be cerebral or obstructive. The former type is nearly always terminal, and the latter usually is. In the former type the vomiting comes on at frequent intervals, and is usually small in amount. The latter is often large in amount, and may become feculent. Both of these types are general manifestations, and have no direct relation to the appendiceal region.

**Tympany.**—The contour of the abdomen in uncomplicated appendicitis is not much changed. Some distention in the region of the head of the cecum may be present, but the general contour is not markedly affected.

Sometimes in the beginning a more marked tympany may be present and it follows closely the vomiting and generalized pain. This early intestinal distention appears when the first reactive hyperemia appears. It is reflex, and not paralytic, in origin. Distended coils of intestine offer a greater surface for the formation of a barrier wall about the point of maximum irritation. It usually diminishes as the phenomenon regresses to the region of the appendix. If alimentary indiscretions are permitted, a fermentation distention may supervene.

In some instances tympany may develop from occlusion of some portion of the gut as a direct local result of the peritonitis. I have noted this once from adhesion of loops of the ileum to an appendix

located in the pelvis, and three times from constricting adhesions in the region of the appendix. The succeeding phenomena are those characteristic of acute obstruction of the gut. These may be confusing since vomiting and distention may be ascribed to a spreading peritonitis. If peristalsis can be seen, felt, or heard, the probability is that there is a mechanical obstruction. Stercoraceous vomiting and collapse should not be awaited.

If the infection spreads, a general diffused tympany may occur in young adults, who usually have tense abdominal walls. Tympany from spreading infection represents a phenomenon of generalized peritonitis, and is considered under the section on general symptomatology. A late tympany may indicate a degeneration of the muscle wall of the gut. Sometimes generalized tympany may be prevented by the extensive plastic exudate which covers the walls of the intestine. When this condition exists in but a portion of the abdomen, tympany may be greatest in that part of the abdomen least affected. In such cases the site of greatest distention may be in the epigastric and splenic region, and it thus may resemble an acute distention of the stomach, when, in fact, the tympany is due to distended coats of small gut which escape around the left border of the great omentum and come to lie over the left border of the stomach.

The border may be retracted instead of being distended, when there is an extreme toxemia or a cerebral complication. This is the familiar scaphoid abdomen.

**Fever.**—The question of increase in temperature in appendicitis is important merely because of its presence. The extent of the temperature increase is of little moment because of the great variability of its range.

Theoretically, an appendicitis may exist without a rise of temperature above normal. Its actual existence is assumed by many writers. Herzog places these feverless cases at 26 per cent, and Rotter at 19 per cent. In such cases it is questionable whether fever did not exist before observation was begun. Murphy emphasized the diagnostic importance of the initial fever, and it will prevent many errors if one holds fast to a belief of its importance. My experience has been that appendicees removed under conditions



in which there is no rise of temperature fail to show microscopic evidence of acute inflammation.

Ordinarily the onset of the disease is characterized by moderate fever, from  $100^{\circ}$  to  $103^{\circ}$  being the average range. An initial temperature above this maximum speaks against appendicitis. Usually the height is reached soon after the beginning of the attack, and runs its course in from three to fifteen days. Herzog classified 139 cases relative to their maximum temperatures. He found a temperature of less than  $39^{\circ}$  C. in 69 cases,  $39-39.5^{\circ}$  in 40 cases,  $39.6^{\circ}$  to  $40^{\circ}$  in 14 cases, and 16 cases above  $40^{\circ}$ . Rostovtseff noted that the highest temperature is observed between nine and ten o'clock in the evening.

Some patients begin with an initial high temperature, accompanied, perchance, by a chill. This type quite regularly reaches a temperature of  $104^{\circ}$  F. or more. Outside of this type, I have rarely observed so high a temperature in uncomplicated cases of periappendicitis. Usually the temperature ranges from  $99.5$  to  $102$ . High initial temperatures should always excite suspicion that the disease is not of the appendix. The development of a secondary abscess is often marked by a high temperature.

When the disease does not extend beyond an involvement of the peritoneum of the appendix, and the peritoneal surfaces coming in contact with it, the temperature may recede to normal within one to five days. If there is considerable induration it may continue for a week or more. It usually recedes by lysis, rarely by a sudden drop; yet many instances of a sudden recession are recorded in the literature. In such cases it is likely that an abscess has already formed which has drained spontaneously into the lumen of the gut.

When abscess formation begins the rise of temperature takes on an indefinitely prolonged course. If the abscess formation remains within the confines of the original adhesions the temperature is not prone to ascend to a great height, usually remaining below  $103^{\circ}$ . If, however, an extension beyond the original confines takes place, particularly if such extension takes place in extra-peritoneal cellular tissue, a much greater height may be reached.

When the initial temperature recedes for a day or more and then mounts again, the development of a periappendicular abscess

may confidently be predicted. The height of the temperature, due to the formation of an abscess, and its subsequent course depend upon whether or not assistance is rendered. When the abscess is drained a more or less sudden drop takes place. An even more sudden drop follows the spontaneous rupture of an abscess into a gut. This sudden drop may also occur when the abscess ruptures into the free peritoneal cavity, attended by the symptoms of shock. When the rupture takes place into the free peritoneal cavity the temperature often goes below normal, but soon rises again. The pulse, it is important to note, suffers in volume and increases in rate.

An abscess left to itself, if of limited dimensions, may be taken care of by the tissues, and ultimate complete absorption will ensue. In such instances more or less uncertainty arises as to the existence of pus. After an abscess has attained a size to be certainly diagnosable, regression will hardly occur until the pus, by some means, gains its liberty. The mere presence of a palpable mass with leucocytosis is not certain evidence of the presence of pus. The mass is more likely due to agglutinated intestines and omentum.

**Pulse Rate.**—The pulse rate is usually increased parallel with the rise of temperature. Early in the disease, when the pains are yet diffuse, the rate may exceed the normal proportion. This is particularly true when nausea and vomiting are present. At this stage the rate is rather more a measure of the nervous state than of the degree of intoxication. Later when the local reaction is in full swing, the normal relations are restored. A pulse rate of from 80 to 120 is usually observed. Often the pulse rate reaches normal before the temperature does.

The often discussed disproportionate increase of the pulse rate is characteristic of a spreading peritonitis. When the infection spreads without limitation the rate may exceed the ability of the surgeon to count. A descending temperature with a rising pulse rate is an omen of the greatest gravity.

The character of the pulse is one of moderate excitability. Early it may be full, even presenting a suggestion of dicrotism. Later it assumes a progressively quieter tone until normal is reached. If it ascends in rate, or lessens in quality, once having regressed, a spread of the disease is suggested. As abscess forms, too, the

rate increases, and may reach 120 or more, particularly if the abscess has reached cellular tissue.

The extremely rapid and thready pulse is characteristic of generalized peritonitis and is observed in terminal stages only.

**Leucocytosis.**—The interest in the occurrence of leucocytosis in appendiceal inflammations is heightened, for it was in relation to this disease that Curschmann made his observations which resulted in the general recognition of the relation of leucocytosis to acute inflammatory lesions.

In a general way the increase in leucocytes runs parallel with the degree of infection and with the temperature. The usual range is between fourteen and eighteen thousand, and with the formation of abscesses the increase may be double these figures. The higher figures may be reached, as noted by French, when extensive abscesses are formed. As localization takes place the count falls, only, like the temperature, to rise again if abscess formation spreads.

It was early noted that the polynuclears undergo a disproportionate increase. Kuttner and Federmann have studied this relationship carefully. The relative increase in the polynuclear leucocytes may reach 90 per cent and more, and the count is apt to be particularly high in cases in which there is extensive exudation into the appendix and surrounding tissues. This disproportion becomes equalized as the intensity of the infection lessens. In some very virulent infections the leucocytosis may not only be not increased, but actually lessened. The polynuclears in such conditions suffer actually and relatively. This leucopenia may be very pronounced in the rapidly fatal types of spreading peritonitis. I have seen the white count as low as 2,300, with 60 per cent polynuclears.

On the whole the leucocyte count is more apt to mislead than to aid in so far as the determination of the severity of the attack goes. It is of some value in differential diagnosis when typhoid fever or tuberculosis is suspected.

### Diagnosis

Appendicitis is usually characterized by symptoms that are typical. When generalized abdominal pains, with or without nausea present, followed by tenderness in the right flank and fever, the

appendix probably is inflamed. Appendicitis is less often overlooked than other diseases are mistaken for appendicitis. Taken in the aggregate this organ is more often the subject of misdiagnosis than any other abdominal organ. The diagnosis of appendicitis involves not alone the site of the disease, but also its character. This is important to remember in the diagnosis of the disease of any organ so inconstant in its position. But, since the evidence available is due only in part to the characteristics of the lesion it produces, quite as much weight must be placed on the distinctive characters of the diseases which simulate it. After the positive signs of the disease have been carefully considered, diagnosis by exclusion should always be carefully considered.

The variations from the typical cases are numerous. Mild forms of the disease may be limited to colicky pains, at first diffuse, but later localized, or the localization may be absent. Rise of temperature may not be detected, and muscle rigidity and tenderness will not be noted unless searched for by a competent diagnostician. In children the entire attack may be represented by generalized pains with vomiting, possibly with an evanescent rise of temperature. The nature of these attacks may not seem clear until more serious changes in the appendix precipitate peritoneal involvement. In the community where I did general practice and have since seen much of the graver diseases, I have observed that the youngsters who were subject to bellyaches twenty years ago have most of them developed appendicitis in late years.

The chief source of error in the diagnosis of appendicitis is the attempt to reach conclusions without adequate evidence. This lack of data may be due to failure to carefully study the patient, or to the fact that the evidence has disappeared. The latter state may be remedied in part by a carefully recorded history. The advent of pain, its time relation to the taking of food, its intensity as measured by the subsequent acts of the patient rather than by the adjectives that are used in describing it is what counts. If fever is said to have existed it must be determined whether the opinion is based on thermometric measurement. If the patient's physician states that muscle rigidity was present, his ability to determine this point must be taken into account. Repeated examinations are

desirable if this is possible, and when the chronic type is in question this is imperative.


When the probable diagnosis of appendicitis is arrived at, careful consideration must be given the question as to whether an inflammation of the appendix could give rise to the symptoms complained of. For instance a history of pain in the region of the appendix associated with occipital headaches or right subscapular pain should at once convince the examiner that an appendicitis could not explain these phenomena. Every abdomen should have painted across it the familiar railroad sign—stop, look, listen.

**Differential Diagnosis.**—In all but the more typical cases the diagnostician must consider the possibility of the existence of other conditions which simulate periappendiceal lesions. The range of possibility is large, and each of these may require an analysis in concrete cases. It is only by exercising every care that errors can be prevented from creeping into the experience of even the most careful diagnostician. With this idea in mind the various diseases which require consideration may be presented in order.

**Kidney and Ureteral Colic.**—Pain due to the passing of a foreign body along the ureteral tract may be located in the region of the appendix. It may be severe in character and radiate to the epigastrium or umbilicus. Vomiting is often present; slight fever and rectus rigidity may be present. This picture resembles so closely an attack of early appendicitis that factors specific for the urinary tract must be sought. Pain radiating to the bladder, perineum, or testicle suggests a urinary lesion. Deep tenderness over the kidney may be a bit of added evidence. A history of pain brought on by jarring of the body, formerly much depended on, is now superseded by the more positive evidence supplied by the x-ray. The kidney may be enlarged and palpable and sometimes tender. The urine may show blood, but this is sometimes present in appendicitis. The leucocytes in the urine likewise may be increased both in kidney stone and appendicitis, and only when abundant do they present fairly reliable evidence in favor of stone. The x-ray may show a stone, which is quite conclusive, but an appendicitis may exist in the presence of a quiescent stone, as I once observed to my chagrin. The presence of a stone with associated lesions of the urinary organs, the direction of the radiation of the pain may aid in preventing

such an error. The muscular rigidity, when present in kidney colic, is not so pronounced as the degree of pain would indicate were the pain appendiceal in origin.

**Diseases of the Gall Bladder.**—Gallstone colic and cholecystitis, due to stone or other causes, sometimes produce symptoms which simulate appendicitis. When the colic is typical, the subhepatic or epigastric pain, radiating to the back or right shoulder, presents a picture fairly typical, especially if the patient be "fat, fair, and forty." The pulse is little affected, and the temperature but slightly disturbed. When a cholecystitis supervenes, the muscle rigidity is confined to the upper part of the right rectus. A tumefaction just below the costal border, moving with respiration, when present, is typical. When there is no tumor, a deep tenderness may be present. The history of previous attacks is of importance particularly when attended by jaundice. Sometimes the gall bladder lies very low, and when inflamed may simulate an acute appendicitis. I once saw an abscess pointing in the ileocecal region which when opened discharged many gall stones. Conversely, an appendix lying lateral to the cecum and extending to the liver may simulate a pericholecystitis. As an example may be mentioned a case of a man of fifty years who had had several attacks of pain situated just below the costal border. There was marked muscular rigidity, and the pulse and temperature indicated an acute infection. He was seen in one of these attacks by a distinguished internist, who confirmed the diagnosis of cholecystitis. At operation an appendix the size of the finger lay lateral to the colon, reaching to a point just lateral to the gall bladder. There was tenderness and rigidity of the anterior border of the quadratus lumborum muscle at its upper end. This point should have caused me to suspect the appendix rather than the gall bladder.

**Perforating Ulcers of the Stomach and Duodenum.**—When ulcers of the pylorus or duodenum perforate, a severe pain is set up, which is sometimes mistaken for an acute appendicitis. The initial pain in ulcer is epigastric, attended at once by rigidity. The characteristic feature of an ulcer is the great intensity of the pain.  There is probably no pain more severe. The most expressive adjectives in the language are used in succession to make known the feelings of these victims. The French have employed the word

Ah!  
Ah!

“brutal” to characterize this pain, and were it the product of a free will it might be so designated. Equally characteristic is the definite time of onset. Patients state the exact time of the day at which the pain began, and not infrequently indicate the very act they were engaged in when the pain began. One patient mentioned the fact that it was just as he stooped to pick up a shovel; another had reached over from his chair to pick up an object from the floor; another had just arisen from the dinner table. Along the same line, (but less definite), was the assertion of a young student that the pain began while he was kissing his sweetheart good night. In the early stage the tenderness is limited to the upper abdomen. Later there may be pronounced tenderness lateral to the colon and in the ileocecal region. This is readily understood when it is remembered how quickly the gut contents reach this region by flowing over the great omentum. Sometimes the amount of fluid collecting here may be so great as to be demonstrable by physical means.

The history of epigastric disturbance is usually given as of importance in diagnosing a perforated ulcer. Often unfortunately the patient fails to recall any previous epigastric disturbance. The pulse may become rapid early and the temperature may be sub-normal.

Sometimes the region of the impending perforation is partly walled off before the disaster actually occurs. In that event a localized abscess may form in the region of the ascending colon, and it may then resemble an appendiceal abscess.

Gangrenous appendicitis also sometimes causes the most excruciating pain. I have not seen this except in appendices which became wholly gangrenous from thrombosis of the supplying arteries. The pain is generally not located in any given point, as it is in ulcer, and the pulse and temperature may be but little disturbed. When some time has elapsed since the advent of the pain improvement may have occurred in the appendicitis while in a perforated ulcer the symptoms continue unabated.

When an appendix perforates with the escape of gut contents the course may be as stormy as in perforated duodenal ulcers. Here history of previous attacks and location of the initial pain in the region of the appendix may aid.

When the surgeon is confronted by such a grave crisis as either of these conditions presents, no time should be lost in diagnostic niceties. A right rectus incision will reveal the presence of duodenal or stomach contents free in the peritoneal cavity or an appendix obviously the source of the irritation. The more courageous surgeon will make the incision over the most likely source of trouble and if he is wrong will abandon this incision and make a new one in accordance with the revised diagnosis. This leaves a permanent record, written in scars, of the error of diagnosis, but it gives the surgeon the better field which results from a correctly placed incision.

**Acute Pancreatitis.**—Acute affections of the pancreas may simulate acute appendicitis because of the generalized pain and abdominal distention. This pain, like perforating ulcer, is extremely severe. It is situated in the epigastrium and may radiate straight through to the back. It usually occurs in males at or beyond middle life. Distention, vomiting, and other symptoms of obstruction begin early. It is the epigastric fullness and the general evidence of abdominal distention, in conjunction with the above symptoms, that points most to the diagnosis. *Cyanosis! also.*

**Diseases of the Urinary Bladder and Rectum.**—When the appendix hangs over the brim of the pelvis it may produce a pelvic peritonitis, but when the organ is long enough to reach the bottom of the culdesac or to come in contact with some organ a localized abscess may form. When the appendix lies entirely in the true pelvis no symptoms may be produced in the iliac fossa. In some instances all the symptoms are referable to the true pelvis and its contained organs.

The symptoms produced in such cases are usually referable to the bladder or the rectum. Sometimes vesical tenesmus or even retention may initiate the complaint. A boy, aged 12, without other symptoms was unable to urinate. He was catheterized without difficulty by his physician, and this proved the urethra free and the urine normal. When examined, he had a temperature of 100° which was not noted before vesical distention became prominent. The abdomen was soft and nowhere sensitive. Bimanual examination showed a mass behind and to the right of the bladder. Operation showed an appendix adherent by its tip to the latero-



posterior surface of the bladder, imbedded in a mass of exudate. This type is seen most frequently in young boys.

I have seen large abscesses form with but little marked symptoms. The very paucity of symptoms associated with retention is presumptive evidence of a pelvic appendicitis; later, rise of temperature always supervenes and usually there is physical evidence of an abscess. The retention is usually due to irritation of the sphincter, and not to direct pressure. I have never seen complete retention in an adult in the presence of large perivesical abscesses.

Abscesses in the pelvis by irritating the rectal wall excite a production of mucus, often of pain and tenesmus. A sudden profuse production of mucus, particularly if the mucus is streaked with blood, presages a rupture of the abscess into the rectum.

Sometimes the chief symptom, aside from disturbance of the bladder, is a diffuse tympany. This is most likely to be the case when there is extensive involvement about the rectum, or when coils of small intestines are involved in the formation of the walls of a localized pelvic abscess. I once operated on a patient presenting the symptoms of intestinal obstruction without discernible cause. Four coils of ileum dipped into the pelvis to surround an inflamed appendix plastered in the floor of the culdesac.

**Typhoid Fever.**—Slowly beginning inflammations of the appendix may simulate incipient typhoid fever. This is likely to be the case when the appendix lies lateral to or behind the cecum, and particularly if it is primarily retrocecal. The explanation of the absence of the cardinal symptoms of appendicitis is easily understood when we remember that this type represents essentially a cellulitis of the retroperitoneal tissue, and not a peritonitis. Local tenderness and the constitutional evidence of infection are all that is apparent. Litten's method of determining the presence of this deep edema was to pick up a large fold of skin, first on the affected side then on the other. If edema is present the fold so picked up will appear to be more voluminous. A deep edema of the lumbar muscles, usually with tenderness, is the most reliable sign of impending abscess in this region. The temperature early in the disease is often higher than is usual either in the intraperitoneal location of the appendix or in typhoid fever. The pulse is apt to be rapid and wiry rather than dicrotic, as in typhoid. There

is usually an absence of rigidity of the recti muscles in retrocecal appendicitis. The leucocyte count is often distinctly increased. I have seen it as high as thirty-five thousand. A case in point is as follows:

A male, aged 35, complained of malaise and an uncomfortable feeling in the right side with anorexia. When first observed by his physician on the fifth day he had a temperature of  $103^{\circ}$ . A continued fever with abdominal distention followed for the next three weeks. Failing to develop the Widal reaction or to show distinctive clinical signs of typhoid he was brought to the hospital. He had at that time a deep edema over the quadratus lumborum muscle with deep tenderness. There was marked general distention. The blood count was as indicated above. Incision disclosed an appendix with much thickened walls lateral to the ascending colon and imbedded in cellular tissue which was infiltrated with pus. Massive drainage brought prompt improvement, but he died of a pulmonary embolism three weeks after the drainage.

The recognized signs of typhoid rose spots and the Widal reaction are absent in retrocecal appendicitis. Conversely, typhoid fever may resemble appendicitis. Sudden pain in the region of the appendix with tenderness in the iliac fossa is sometimes observed. The error may not be discovered until the definite signs of typhoid fever develop, perhaps after the appendix has been removed.

I well remember a lad of fourteen who, after a hearty meal, was taken with acute pain in the ileocecal region. Within a few hours the temperature had reached  $103^{\circ}$ , and there was deep tenderness over the cecum. There was some voluntary muscular rigidity. The pulse was full, bounding, and not over eighty. The following day it became dicrotic. The muscular rigidity disappeared, and distinct ileocecal gurgling could be elicited. The Widal reaction was not positive for ten days. Hemorrhages occurred repeatedly during the third week, and he died during the fourth week with intestinal perforation.

This case illustrates the chief differentiating factors. The muscular rigidity lessened when the patient's attention was attracted elsewhere. The muscular rigidity had a swinging comeback, and not the tense guarded hardness of acute peritoneal irritation. The

pulse was characteristically typhoidal. The leucocyte count in this case was eight thousand a few hours after the onset. The leucocyte count is very misleading early in the attack. Later it becomes of more certain import. If distinctly pronounced, say fifteen or twenty thousand, particularly if the polynuclear count is high, it gives evidence in favor of the suppurative lesion. In the sudden onset of typhoid I have seen white counts as high as twelve thousand. In following up the count, however, the typhoid count will decrease while the count in suppuration is more likely to increase. Usually before the leucocyte count can be followed long enough to give evidence of the cause, definite evidence will be available from other sources.

**Female Sexual Organs.**—Numerous diseases arising from the adnexa may simulate a periappendicitis. In some of these the resemblance is so close that the most painstaking analysis may fail to produce a positive diagnosis. In some rare instances, even all the evidence obtained from the operation and the pathologic laboratory put together may fail to make a positive differentiation. It is desirable to consider all the physical evidence before the history is taken into account. This is desirable since appendicitis is a thoroughly respectable disease, and young females in giving the history may avoid factors that are not compatible with the most exemplary social conduct.

I have found this the most difficult condition to differentiate from appendicitis. Pain in the right groin in young women if more or less persistent is likely not caused by an appendicitis. If the pain radiates over the hip or down the thigh the pain most certainly is ovarian in origin. Unless a history of initial epigastric pain, with or without nausea, preceding the groin pain is obtained, the appendix is not involved.

The various diseases that may simulate appendicitis or its complications will be considered separately.

**Ectopic Pregnancy.**—The sudden pain of extrauterine pregnancy when located on the right side, may simulate appendicitis. The pain is usually more severe than in appendicitis except in the gangrenous types. The pain in tubal disease is usually situated low in the pelvis often associated with vesical or rectal tenderness. The pain is often described as bearing down. Collapse when pres-

ent indicates tubal trouble, while vomiting points to appendicitis. Anemia when marked makes tubal trouble nearly certain. A leucocytosis above fourteen thousand indicates appendicitis. An initial rise in temperature speaks for appendicitis while a temperature of several degrees is usually present after a day or two when there is a blood clot in the culdesac. An initial low temperature and rapid pulse operates for tubal disease. Physical examination in the tubal disease shows moderate rigidity of the lower segment of both recti muscles, often with but moderately deep tenderness. The vaginal examination may show a tubal lesion or a roundish, fairly hard bloodclot palpable in the culdesac. If the social position is such that a careful history is permissible, the typical relation of the attack to a missed period may be a great aid; but when it is not, the statement of the patient is more apt to mislead than to aid. In cases where there is doubt, delay may aid in solving the problem.

A tubal pregnancy may cause pain and simulate an appendicitis in the groin before rupture. Physical examination will demonstrate a thickened tube and an absence of muscular rigidity.

**Parametritis.**—Infections of the parauterine connective tissue resulting from abortion may give rise to symptoms of pelvic infection simulating inflammation of the appendix located in the pelvis. The pain may be referred to the iliac fossa, and infection may extend over the pelvic brim, simulating a tumor mass originating from the appendix in this region. If a history of abortion can be obtained and a vaginal examination be made, the extraperitoneal character of the exudate becomes apparent. The presence of a hard, bone-like mass situated over the body of the ischium indicates unmistakably in favor of a parametritis. If one can separate such a patient from her friends, and secure an examination under an anesthetic, these facts can be determined with accuracy.

**Ovarian Tumor with Twisted Pedicle.**—When the pedicle of an ovarian tumor becomes twisted the wall suffers from lack of nutrition and becomes an irritant to the surrounding peritoneum. The symptoms are those of intense abdominal pain, usually located in the pelvis and lower abdomen. When the previous existence of a tumor is known the diagnosis is easy. Even when not known, if a vaginal examination is made, the rounded mass can be

outlined, which is quite unlike the exudate from appendicitis, particularly of a beginning appendicitis. After the disease has existed some days the tumor may be obscured by the surrounding exudation. Temperature and leucocytosis may closely simulate the findings in appendicitis, save that the initial rise is apt to be greater in case of the twisted pedicle. There may be a history of repeated attacks, which simulates very much the course of appendicitis. The muscular rigidity is usually bilateral even though the cyst is small and lies in one side of the culdesac.

**Gonorrheal Perisalpingitis.**—The differentiation between an acute appendicitis and a gonorrheal infection of the tubes often presents difficulties because of the position in which the consultant is placed. Openly to suspect such an infection would excite sedition. In such instances the manner in which the patient answers questions, her disposition to observe carefully the movements of the examiner, the general attitude of apprehension, much like one undergoing a Bertillon measurement, should cause the examiner to exercise the greatest caution in expressing an opinion.

The history may give additional aid. The first question may bring forth a profusion of information, particularly as to the causation. A patient once told me the pain was caused by lifting a bucket of water, and I believed it. Often the trouble is ascribed to a fall, which perhaps conveys more truth than the patient intended. An attempt is often made to mislead the examiner as to the site of maximum pain. The region of the appendix is often indicated in order to throw the examiner off the track. I once met such a situation by directing that the patient be allowed to place half a mustard plaster over the site of maximum pain. Returning later I found the site of hyperemia following its use directly over the pubes. After her sister had gone to the corner drug store and her mother to the kitchen, the patient readily told all about her trouble. When not hampered by the presence of fastidious relatives the diagnosis is not so difficult.

When only the right tube is involved the pain may be referred to the right groin and when sudden in onset, accompanied by vomiting, it may simulate appendicitis very closely even when all the factors are available. Though the tubal infection be unilateral the lower portion of both recti will be rigid because the entire pelvic

peritoneum responds to the irritation. In appendicitis the right rectus alone is rigid unless it lies in the pelvis and has caused a generalized pelvic peritonitis, when both recti may be rigid. In such instances the right rectus will be found to be rigid for a greater extent than the left.

The temperature is prone to be higher in the tubal infections. Temperatures of  $103^{\circ}$  or  $104^{\circ}$  or more, are often reached early in the attack. Such a degree of fever would be very unusual in beginning appendicitis. Vesical and rectal tenesmus may be pronounced in tubal disease. Leucocytosis is more apt to be high early than in appendicitis, though this evidence should not be considered until after the diagnosis is made.

When a true history can be obtained the previous existence of a leucorrhea, or vesical irritation, or a prolonged menstruation may aid. When a physical examination is possible, and the microscopic demonstration of the gonococci is made and palpation reveals a pelvic mass or a thick tube, the diagnosis is easy. One must remember, however, that the diagnosis of a gonorrheal salpingitis only is made by such evidence, and appendicitis is not excluded thereby. I am led to emphasize this point because I once had a patient who was known to have chronic gonorrheal tubes. An acute attack of pain in the right side was ascribed to a harmony meeting among the organisms of the tube. A belated operation showed an appendix perforated near its base. The versatility of modern civilization must ever be kept in mind.

The site of pain in the normally located appendix is higher and more lateral than that of tubal disease. Tubal pain is apt to be greater just over the pelvic brim. Sometimes in appendicitis pain may be elicited by pressing deeply over the anterior edge of the quadratus lumborum muscle, while a painful tube is not aggravated by this maneuver. In tubal disease the opposite side is usually tender to deep pressure. Bilateral or left-sided tubal disease will seldom give rise to difficulty in diagnosis, but the possibility of an unusually located appendix should not be forgotten. I once found an appendix in an inguinal hernia on the left side.

The difficulty in differential diagnosis is further increased because of the frequency with which the tubes and pelvic peritoneum are involved in appendicitis. I have made it a point when operating

on women for appendicitis so to place the incision that the pelvic organs can be explored. In fully 10 per cent of cases the presence of a distinct perisalpingitis is found. In some of these instances, at first glance the primary focus of infection may not be apparent. The point which permits of a differentiation is chiefly that in salpingitis secondary to appendicitis the mucosa of the tube is not involved, while in gonorrheal salpingitis this is of course the primary seat. The occlusion of the tube is often stated to be a sign of endosalpingitis. My studies lead me to believe otherwise. The inner layer of the fimbria is the fixed point, and when a subserous edema, from any cause, occurs, the end of the fimbria becomes turned in. In appendiceal perisalpingitis the tube is not densely infiltrated, nor is it so apt to be elongated as in the specific disease. Conversely, in primary tubal disease the appendix may be involved. Fortunately, one need feel no compunction against the removal of the appendix when it is involved. Examination will show foci of infection if it is the primary source, and a diffuse periappendicitis if it is secondarily affected.

When a generalized pelvic peritonitis has resulted from a perforated appendix, the entire thickness of the tube may become involved. In such a case the lumen of the tube itself may contain pus. When the walls are thickened as the result of a perisalpingitis, they are less edematous than in gonococcal infection. This is due to the pronounced hyperemia of the submucosa. On section there are fewer cells than in the gonorrheal tubes, and there are proportionately more polynuclear leucocytes.

**Ovarian Hemorrhage.**—Hemorrhage within the substance of the ovary may give rise to severe pain in the lower abdomen, but is unattended by fever of any considerable magnitude or by leucocytosis. Muscular rigidity is absent. The character and location of the pain simulate tubal abortion rather than appendicitis, but it lacks the constitutional disturbances of a ruptured ectopic pregnancy.

**Dysmenorrhea.**—(Noninflammatory pelvic complaints.) Many appendices are removed because of pelvic disturbances other than those caused by frank inflammation. The source of these pelvic pains is not known, and here it suffices to separate them from inflammations of the appendix. The problem is simplified if it is remembered that dis-

turbances from this organ are confined to those due to reflex disturbances, transmitted usually to the epigastric region, and those due to periappendicitis which are local in nature. Constant pains in the groin are almost certain not to be caused by any disease of the appendix. The factors which point to their source in the pelvic organs is their dragging character, often associated with sacral pain. Often they radiate over the hip or down the course of the obturator nerve. They recur at frequent intervals, the patient often declaring that they are constant. Not infrequently the pains are aggravated by menstruation, less often they are relieved by the monthly flux. In a previous publication I attempted to clarify this problem by the use of French expressions. The one type, characterized as "hyperovarie," is represented in the robust girl with an abundance of animal vitality who does not complain of pain when there is active physical exercise in prospect. The other type, the "hypoovarie," are generally frail and slender, and are possessed of limited physical ambition. They move, as George Ade says, as if they were on casters. This type have small furrowed ovaries, and time but confirms their complaints, while the first mentioned type lose their pain as soon as they engage in the gentle art of pushing a perambulator.

The hyperovarie type are not injured by a needless appendectomy, while the other type are apt to suffer a permanent nervous upset. They complain of adhesions, and clamor for repeated operations.

It has always been my rule not to remove the appendix from a young woman unless there is physical evidence of a periappendicitis or a distinct history of vomiting, fever, and localized pain.

**Pyelitis of Pregnancy.**—Not infrequently there occurs an infection of the kidney in pregnant women attended by bacteremia, sometimes pyuria. Sometimes there exists merely an infection of the pelvis of the kidney sometimes an infection of the kidney itself. The characteristic symptoms are relatively high fever, often initiated by a chill, sometimes attended by psychic disturbances. There may be pain on pressure in the appendiceal region. Since this complication occurs usually in the later months of pregnancy, physical examinations are rendered more difficult. The diagnostic features are the deep



tenderness over the kidney and the absence of muscular rigidity over the site of the appendix. I once had a patient with a suppurating appendix which lay over the lower pole of the kidney. Since appendicitis in pregnancy is of greater moment than in the nonpregnant, exploration seems to be the only means of solution in such cases.

**Genital Infections in the Male.**—Epididymitis and diffuse infection may give rise to pain referred to the region of the appendix. There is an absence of muscular rigidity, except possibly over the lower abdomen just above the inguinal canal. The epididymis is tender, and obviously the site of an infection. Leucocytosis and rise of temperature may be as marked as in appendicitis. The testicular lesion is sometimes so little obvious that its presence is not suspected.

**Hernias.**—A beginning inguinal hernia may cause pain in the groin, sometimes higher. These are particularly apt to confuse when they split the muscle layers and extend upwards towards the appendiceal region. These may become irreducible and inflamed, thus heightening the simulation. A bit of omentum may become adherent, thrombotic, and inflamed, and thus produce a tender tumor above Poupart's ligament.

A double difficulty is encountered if an appendix lying in a hernial sac becomes inflamed. The correct diagnosis is seldom made (just twice in sixty-two cases to be exact,—Wassiljew). In one of the cases I observed, the diagnosis should have been made. A large inguinal hernia which had not been reduced for many years suddenly became violently inflamed. There were no symptoms of strangulation. Fortunately, I made no attempt at reposition, but proceeded at once to operation. A perforation at the base of the appendix had already occurred, and a prolonged suppuration in the sac resulted. In my other case the symptoms of strangulation were primary. The cecum and a loop of ileum showed evidence of strangulation. I suspect the appendix became inflamed because it suffered the vascular embarrassment of strangulation of the gut higher up. There was a general perivascular infiltration with no local foci.

**Diseases of the Chest.**—It is generally recognized that in young children pleural pains are frequently referred to the abdomen. It is not so generally appreciated that the same condition may

prevail in adults. In children the question as to the presence or absence of muscular rigidity is answered with difficulty. The respiratory excursions are limited in both diseases, but in primary pulmonary diseases they are more frequent. The diagnosis is dependent largely on the demonstration of a pulmonary or, at least, an intrathoracic lesion. This is often difficult, for some days may elapse before definite physical signs develop. In such instances a more tympanitic note on the right side may put the surgeon on his guard even before there are any auscultatory changes. In adults the physical signs are usually present, and found, if carefully sought. Error here nearly always comes from lack of diligence, though it must be added that all physical signs may be absent in the first few hours. In the adult, muscular rigidity can be demonstrated in all patients demanding immediate operation.

Possibly a too rigid attention to the pulmonary signs causes many cases of associated infection of the peritoneal cavity by the pneumococcus to be overlooked. This is a matter of small practical moment, however, because a pneumococcic peritonitis is not a subject for early operation, while the removal of a normal appendix in the presence of pneumonia is embarrassing to the operator, and places a serious additional burden on the patient.

### **Chronic Appendicitis**

The following interpretation of the term *chronic appendicitis* is an attempt to correlate symptomatology and anatomic findings. It is desirable that the surgeon know whether or not the organ he holds in his hand is responsible for the existence of the symptoms for the relief of which he is operating. There are few more abused terms in surgery. The vaguest local or general symptoms are too often taken to warrant the removal of the appendix and the recovery of the patient from the operative traumatism is accepted as vindication of the opinion. Much has been written by surgeons and by pathologists, and yet more by those who are neither. There has been but little attempt to correlate the histologic with the clinical findings. This discussion is based on such a study. The various conditions discussed under this head from time to time do not admit of ready classification yet certain indefinite groups may be constructed.

The following groups may be recognized: Those cases in which recovery is complete, those which never come to an acute attack, and finally, the vast group in which the appendix is not diseased at all.

**Remittent Appendicitis (Postappendicitis, Fenger).—**A designation for this group is as difficult as forming a characteristic term for tumors, which it will be remembered Virchow stated could not be made. It is meant to include under this head those cases only in which, after an acute attack, recovery is not complete and a



Fig. 186.—Remittent appendicitis. This patient had a sharp acute attack and was operated on in the free interval. An adhesion of the omentum with slight interstitial changes is all that remains to indicate a past inflammation.

series of symptoms continue either as a continuance of reactive processes or from conditions resulting therefrom. Fenger designated the first group "postappendicitis" (Fig. 186). The second group presents residual processes such as adhesions, etc. In very rare instances these remain as anatomic structures, interfering mechanically with the function of the gut (Fig. 187), or in sclerotic processes within the appendix itself the significance of which can only be abstracted from the subjective manifestations of the patient before and after operation.

From the first group must be excluded the ordinary everyday appendicitis patients who suffer acute attacks, and recover, only to be affected later by a renewed attack. Sonnenburg applied the term *chronic appendicitis* to this group, obviously not without justification, for it can not be determined with certainty whether or not the recovery has been complete in the interval. Possibly relapsing appendicitis would be a better term. Be this as it may, the

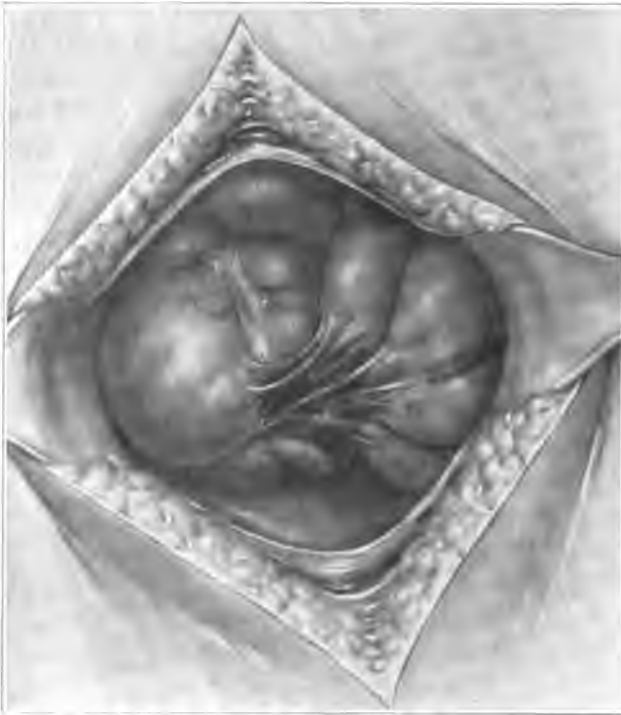


Fig. 187.—Adhesions of several loops of ileum about the cecum. There had been a perforation about the base of the appendix. Intermittent symptoms of intestinal obstructions persisted.

diagnosis of these cases is usually easy from the history alone, and appendices removed in the interval may present no anatomic evidence of past disease. An appendix which upon removal shows no evidence of disease can not, in the light of present knowledge, be said to be the cause of symptoms, though we must admit that, in the light of studies in infections from other sources, such a rela-

tionship may exist. Therefore, it seems as justifiable to exclude these from the group of chronic diseases, as in the case of the kidneys or tonsils in which acute attacks may present themselves from time to time without their previous state being called into question by the terminology.

**Chronic Productive Appendicitis.**—The elemental factor in the determination of this group is the history of an acute attack. This may be clear from the recounting of the cardinal symptoms, possibly confirmed by the attending physician who observed the patient at the time of the attack. More often the history is less clear. Sometimes one must look back to the period of childhood when attacks of indigestion and vomiting and general abdominal pain occurred. It has been a source of unending astonishment to many to observe with what regularity children seen many years ago with these complaints, return in after years with unmistakable appendicitis. So often has this observation been made that the syndrome of acute abdominal pain with vomiting and fever has come to mean to me only acute appendicitis. Green apples may produce pain and vomiting, but not fever, nor does the encounter with the first "plug" or cigar. These early histories are important, but must be elicited with skill, for a positive history may be forced from most persons. When spontaneously presented such a history gives a basis for the consideration of the possibility of the existence of chronic changes within the appendix incident to an acute attack.

*Pathology.*—The appendix may appear thickened and in a state of abnormal rigidity. The finger, in transporting the organ into the wound, may note this more clearly than the eye. The organ may present an abnormal translucence, due to an absence of deep capillary circulation (Fig. 188). Perhaps a few superficial vessels may make up for this deficiency of the deep vessels by undue prominence. More often the superficial vessels about the head of the cecum and the mesentery of the terminal ileum show a marked prominence. Sometimes an enterolith is palpable, and sometimes a scar; or a distention due to soft accumulations of feces and mucus may be noted.

The microscopic changes are those of chronic reaction. Aschoff has well designated this state as "appendicitis cicitrans retardata."

Any of the changes belonging to this state may be noted. There may be proliferation in the lymph follicles (Fig. 189) or round cells may be scattered about in the connective tissue in the region of the follicles. Round cells or leucocytes may be seen between the gland



Fig. 188.—Chronic induration of the wall of the appendix. The macroscopic appearance was little altered but the organ was dense to the feel because of the increased amount of fibrous tissue.

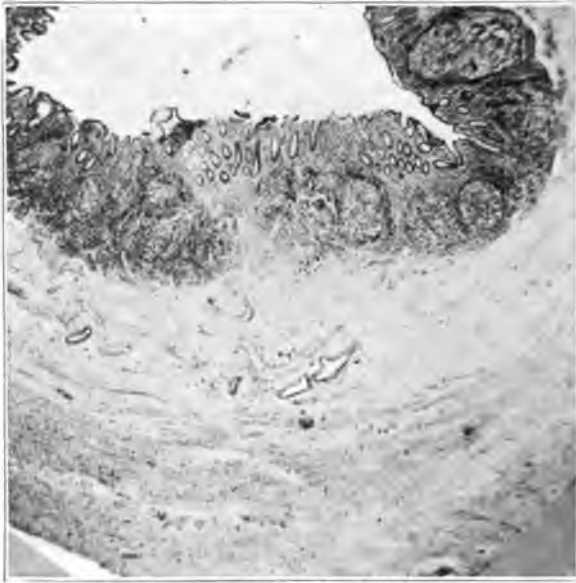


Fig. 189.—Chronic appendicitis in which the lymph follicles remain prominent, simulating hypertrophy of the tonsil. The vessels in the submucosa show plasma cell infiltration.

cells. The gland cells may show a cicatricial deformation. The muscle layer may stain less intensely, and the muscle fibers may be pressed apart by a myxedematous or pseudomucinous exudate (Fig. 190). The subserous and submucous connective tissue each may show

a lessened affinity for acid dyes, and the exudate between the fiber bundles may press them apart. The subserous plexus of vessels may show an intense endarteritis, even to the point of obliteration.

Any or all of the anatomic changes above recorded may be present. Without careful examination with the use of various dyes the changes may escape notice. Because of the difficulty of distinguishing nerve fibrils from elastic fibers any change in the former can not be determined with certainty. This is made more dif-

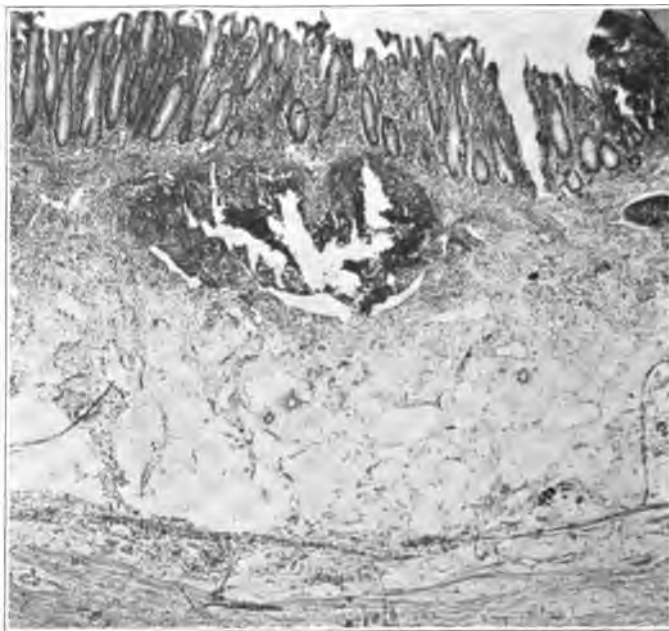


Fig. 190.—Section of a subacutely inflamed appendix. The submucosa is much thickened due to a pseudomucinous exudate within the connective tissue spaces.

ficult by the fact that the elastic fibers are increased in number in chronic appendicitis.

*Symptoms.*—When acute exacerbations occur, reflexes may be instituted. The lesser degrees of reaction may act the same way. The reaction may be great enough to excite a local tenderness without spontaneous local pain, or spontaneous local pain may be caused without local tenderness. The latter align it with a recurrent acute appendicitis self-evident in character, and need not

be considered here. The chronic reaction not sufficient to produce spontaneous local pain may give rise to gastric disturbances. In such instances reference to the appendix can be arrived at only by exclusion. If local pain elicited by pressure is present a connection may be inferred. An association is more likely if the history of an acute attack is obtained.

The manner of the production of the gastric disturbance is not certain. It may be assumed that the irritation of the splanchnic nerve is transmitted to the semilunar ganglia. Any attempt to say from the appearance of an appendix whether or not such reflexes were present is not possible. Deep tenderness to local pressure exists only when the appendix is rigid, because of the exudate above mentioned. This is constant. Superficial tenderness counts against any local changes. Spontaneous local pain is caused by periappendicitis, as previously discussed in the section on acute appendicitis.

**Postappendiceal Cicatrization.**—In this type of chronic cicatrizing appendicitis there are no signs of active organic processes. All represent end processes.

*Pathology.*—Small cicatricial bands juxtaposed to masses of fat with the mesenteroleum are sometimes noted. It is a question whether these are end results of reactive processes or merely atrophic changes. Personally, I am inclined to the latter view.

The changes undoubtedly produced by inflammation may be grouped into those of the appendix itself and those of its environs. In the appendix itself patches of scar tissue, including the whole wall, including the mucosa, are the most frequent. The destruction may be so great as to produce an occlusion of the lumen for a greater or less extent. Sometimes the destruction has been so complete that a mere band of scar tissue may unite the terminal portion of the appendix to the cecum. In none of these conditions is there any trace of existent inflammatory processes.

The lesions in the environs of the appendix are represented by adhesive bands. These may exist between different portions of the appendix or between the appendix and cecum or ileum or between the appendix and some foreign point, such as the parietal wall, tube, ovary, uterus, or a hernial sac. There is scarcely a region in the abdomen to which the appendix may not become attached.



Bands may extend from one point to another in the region of the appendix, and may limit the movement of some normally movable point, or may, in rare instances, produce an actual constriction of a hollow organ.

*Symptomatology.*—Scleroses within the appendix are often discovered in patients who gave no evidence of their existence. When no limitation of movement is exercised on any neighboring organ there are no symptoms. Adhesions to adjacent organs likewise may exist without symptoms. Bands producing mechanical constriction are productive of the usual symptoms of obstruction.

*Differential Diagnosis.*—A great variety of diseases may present local discomfort and pain. Cecal carcinoma, before palpable tumor formation, can only be suspected by its slowly progressive character and slowly developing stenosis. Actinomycosis is so rare that the surgeon may consider himself fortunate if he thinks of the possibility when the organ is exposed during an operation. Tuberculosis likewise may cause a progressively advancing disorder. Stenosis and tumor formation may precede the development of pain.

**Masked Appendicitis.**—In contradistinction to the recurrent appendicitis, by masked appendicitis is meant an affection of the appendix which never manifests itself in acute attacks or exacerbations. This type includes those rather rare instances in which an organ obviously diseased is associated with or simulates chronic disturbances of one sort or another. The appendix does not manifest disturbances of such a character as to direct attention to itself. In such instances the appendix when exposed at operation is much thickened, and exhibits processes of a hyperplastic nature. These have been well designated by Hiller as “masked appendicitis.” Gussenbauer remarks that the term applies not to the nature of the process, but to the possibilities of diagnosis.

*Pathology.*—In this type the entire picture is that of slowly advancing proliferation. The appendices are large, often as large as the finger (Fig. 191). The organ is rigid on manipulation and firm to the touch. The surface is deep red in color, numerous vessels are visible, and the entire environment may show extensive varicosities. On section the organ is firm to the touch, giving a sensation to the knife more like that of cutting a carrot than of

fibrous tissue. The cut surface is moist and glistening and the mucosa may point out of the lumen.

The microscopic section shows a mucosa infiltrated with round cells, and the germinal cells of the lymph follicles show activity of proliferation in their endothelial elements. The submucosa is drop-sical, and contains many round cells and a few leucocytes. The muscular layer likewise is edematous. The subserosa presents the greatest thickening. The fiber bundles making up this layer are pressed far apart by a serous fluid exudate in which round cells are imbedded. The vessels are increased in number, and the walls are much thickened and are infiltrated with round cells.



Fig. 191.—Large thickened appendix. The walls show pronounced fibrous thickening with abundant round cell infiltration. Pseudotubercles are shown near the tip.

In many of these specimens an acute exacerbation has taken place, and some point presents a proliferation or there are evidences of a less localized acute exacerbation. In such cases large numbers of polynuclear leucocytes are intermingled in the tissue.

*Symptoms.*—The symptoms these cases present are those referred to some distant point, such as indefinite pains in the gall bladder region or some chronic disturbance of the stomach. The former is characterized by subcostal pains, sometimes slight attacks of jaundice, and perhaps nausea and vomiting. The latter usually is represented by hyperacidity or hunger pains. The subjective symptoms are associated with local tenderness in the region of the duo-

denum. Often there is a marked disturbance of the nervous system, which much obscures the general picture.

This indefinite symptomatology is usually not cleared up until, after failure to make a diagnosis, an exploratory operation is undertaken. Search fails to disclose any disease of the upper abdominal tract, and the appendix shows changes of the character already noted.

Sometimes, instead, after vainly searching for relief, the patient develops an exacerbation of his trouble with pain on pressure over the appendix, or possibly spontaneous pain which leads to the correct diagnosis and operation. On the other hand, an acute inflammation with periappendicitis may supervene, presenting the cardinal symptoms of an acute appendicitis. Perforation may even take place. The patient discovers after recovery from the operation that his old symptoms have disappeared. He is then able to make his own deductions.

If we seek the cause of the referred symptoms the explanation must be sought in the reflex through the splanchnic system. The irritation in the appendix affects the local plexus, as the periphery of the appendix is not involved, none of the medullated system is affected, hence there are no local manifestations.

**Pseudoappendicitis.**—Aschoff suggests the term *pseudoappendicitis* for conditions which simulate appendicitis. I like the term because it ridicules the limitations of our diagnostic skill. Litten, speaking of pseudoleucemia, compared the term to that of pseudomillionaire, the latter, according to Litten, being an impecunious individual with grandiose delusions of wealth. So we, in speaking of pseudodiseases, are deceiving ourselves into believing we have diagnosed a disease which does not exist.

**Symptomatology.**—In so-called pseudoappendicitis there is pain, more or less constant, in the right iliac fossa, indefinite epigastric distention, general malaise, the so-called “*dyspepsie appendiculaire*” of Longuet. On the other hand the patient may be ruddy and in the best of general health. More often the symptoms are more of a recalcitrant existent or imaginary lover. Constipation or in extreme cases mucous colitis may be present. Frequently scanty or abundant urine of low specific gravity, or the two alternating, may be complained of. Associated with the pains

in the iliac fossa, sacral pains and pains radiating over the hip and down the thigh may be complained of. Headache, sleeplessness, globus, and any other nervous manifestations may complicate the picture.

Physical examination reveals nothing except tenderness. Pain on pressure over McBurney's point is usually the factor that stimulates the diagnosis. In most patients a superficial tenderness exists, which may disappear on deep pressure, but sometimes deep pressure is required to elicit it. Often a roll of muscle resembles in a way a protective rigidity, but it is always as a roll, and not as a broad protective reaction constantly fixed. When pressed deeply down this may give the sensation of a small oblong body, which, because it occupies the position the appendix sometimes occupies, has been interpreted as being that organ. Personally, I feel that I am doing well to palpate these small organs after the abdomen has been opened. The roll of muscle contracts under pressure of the examining finger, and is evidently reflexly stimulated from the pressure beneath. This painful point may be over the usual site of the appendix or just above Poupart's ligament, nearer the pelvic brim, or near the lower pole of the kidney. In a considerable proportion of cases an equal or less tenderness may be elicited at a like point on the other side with like evidence of muscle contraction. Sometimes an additional point of tenderness is demonstrable in the epigastrium, often over a pulsating aorta.

**Differential Diagnosis.**—With such an indefinite disease picture the range of diseases which may need to be differentiated from it is great. Only a few need be reviewed here.

**Genital Disorders.**—Many young women complain of pain in the groin, usually the right or the right predominatingly. This pain is present more or less constantly, and is made worse by hard work, especially housework, rarely by skating or dancing. It is often worse a few days before the menstrual period. The pain is often described as burning in character, and often radiates to the sacrum or over the hip, occasionally down the region of distribution of the obturator nerve. There is superficial as well as deep tenderness in the right lower abdomen, often in the epigastrium and the left side, as above described. When palpation is undertaken the abdomen may be humped up like a mustang about to receive the

saddle girth; or the examination may elicit alternating giggles and exclamations of pain. On the other hand, the patient may lie with perfectly flaccid muscles permitting deep palpation, evincing but little evidence of pain. These are usually noted in females, single in fact as well as in name.

Another class is represented by women who have borne children. They are devoid of the symptomatic frills above detailed, but usually have backache, often leucorrhea, and frequently headache, particularly of the occipital region and of the vault. These patients usually present obvious lesions of the genitals incident to childbirth or to chronic disease of the uterus. Sometimes there is no obvious genital lesion.

Sometimes a chronic salpingitis wrapped about an ovary may produce such pains as are above noted. These changes may be so slight as to escape the palpating finger. A little shortening of a uterine ligament, perchance a tender ovary, may give the clue.

*Sexual Neurasthenia.*—Males form a large contingent of this class. Their complaint is pain in the right lower abdomen, less often in the left. They often complain of burning on urination, and often get up at night. The whole train of phenomena coincident with this state is usually more or less well pronounced. They have deep tenderness, and, if the examiner shares the acute imagination, he may mistake the tense muscle bundles for a hardened appendix. This type of individual has a peculiar psychology. He works for forty dollars a month, his mustache droops, and he does not play baseball on Sunday.

In females the genital bias of the complaint is usually more pronounced, but in some instances the general features as noted in the male only are noted.

Each of the classes above mentioned may perchance have had his appendix removed. If so, he has adhesions at the site of the operation, and his refrain is in nowise changed from that presented before the removal of the appendix.

The elemental character of the neurosis is the factor which guides to the correct diagnosis. If the patient is neurotic or presents neurotic manifestations during the examination, great caution is necessary. In the solution of such problems errors are bound to creep in. A neurotic may actually have a chronically affected

appendix, and, on the other hand, the most demure may present symptoms not dependent on organic lesions.

### **Treatment\***

The literature on the treatment of appendicitis in all countries shows a curiously parallel evolution. Medical treatment, opening of abscesses, operation in the interval or when diffuse peritonitis is present represents the beginning, while the ultimate stage is represented by early operation on all progressive cases.

**Medical.**—The expectant treatment may consist in the use of an ice bag to control the local pain and vomiting. The withholding of everything by mouth according to the Ochsner plan is to be commended. Laxatives are never permissible under any condition. Once the diagnosis has been made and the patient is headed for the hospital morphine may be given to control the pain, not before.

The object of withholding anodynes until the diagnosis is made is that after the pains have been controlled by artificial means the chief of the cardinal signs is obliterated and the making of the diagnosis is much more difficult. Furthermore, after the pain has been controlled the patient is less likely to listen to arguments in favor of an operation than when he is constantly being reminded by intense abdominal pains.

Once an operation is agreed upon it would be permitting needless suffering to withhold morphine until such time as the operation could be performed.

**Time for Operation.**—When a good surgeon arrives is the proper time for the operation. This represents the fatal weakness in this scheme of procedure. The most variable tyro places himself in the category of the competent. What competent surgeon does not observe these with a degree of compassion! Persons who operate an hour or two on a patient afflicted with appendicitis are a greater menace to the patient than the disease. Murphy's dictum "in quick, out quicker" is of vast importance. Quickness is not measured by the clock, but by the degree of traumatism inflicted. Some operators are so slow that it takes them a long

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\*Those interested in the literature of the treatment of appendicitis will find an excellent resume in Sprengel, *Appendicitis*, Enke, Stuttgart, 1906.

time to inflict a little traumatism. An incompetent man, on the other hand, can inflict more traumatism in a limited time in this region than in any other region of the body.

**Indications for Operation.**—The requirements to justify the removal of the appendix are the juxtaposition of a patient with a diseased organ and a surgeon. An endless literature has been written as to the time the organ should be removed.

In general two types may be distinguished: patients who have had an attack and those who are having an attack.

**Operation in the Interval.**—Those who have once had an attack are likely to have a recurrence. Among those whom I have seen in an attack who were not operated on the vast majority of cases have had a recurrence. The history of several previous attacks makes the indication doubly emphatic. In adults after the age of thirty-five and in children recurrences are particularly apt to be severe.

In patients who have had severe attacks and have had abscesses, particularly if there is evidence of perforation as the escape of an enterolith or the formation of fistula, if the wound heals, the patient is practically insured against future attacks. In these subsequent operations are demanded because of scar hernia and local disturbances of gut function. I have never seen such a case suffer severe recurrences of appendicitis.

**Operation in the Attack.**—All acute cases are surgical from beginning to the end. It was formerly advised that internist and surgeon should work together. The internist has as little business loitering about a patient with appendicitis as he has treating a gunshot wound of the abdomen.

No one can tell particularly in the beginning of the attack what type of lesion will ultimately develop. This is quite obvious from a study of the pathology, for what is in the beginning an endo-appendicitis may perforate and lead to the most virulent general peritonitis. Once the diagnosis is made, the fate of the patient depends very largely on the skill of his surgeon. Most surgeons advise operation as early as possible because it is not possible to distinguish between the mild and the potentially severe cases.

There are cases in which there may be some latitude. For instance, when there is a falling pulse and temperature with a sub-

sidence of the local symptoms of tenderness and rigidity it is permissible to await an opportune time. When there is a lessening of the symptoms even if there is tumor formation it may be permissible to permit a spontaneous subsidence provided the patient is where he can be observed. If some days are required for such tumor formation and if rigidity persists, operation had best be undertaken.

If the disease is on the ascendancy, operation should be done at once. It has been advised that in cases of excessively severe onset a few hours should be awaited so that the patient may recover from the primary shock. These are just the cases which demand operation early.

**Place of Operation.**—There is often a question in acute appendicitis whether the patient should be taken to the surgeon or the surgeon brought to the patient. When within ambulance distance unquestionably the patient should be taken to the hospital. Here only can the surgeon do his best work and the patient is assured the best after care. When it is necessary to convey the patient by train, the matter is somewhat different. There is no doubt that the patient can be safely conveyed, but it is exceedingly trying to both patient and friends. Save for the annoyance it brings the surgeon, no doubt, the most convenient way is to call the surgeon, but the safest way is to take the patient at once to the surgeon. The chief danger the patient undergoes in being treated in his home by those not familiar with the after care of such cases is that he will be overtreated. Food and laxatives, from the joint action of friends and anxious doctors, often spell intestinal paresis.

**Type of Operation.**—The type of operation depends, in the first place, on the capability of the surgeon, and, in the second place, on the nature of the lesion. It is possible to individualize somewhat so far as the type of disease goes, but it is not possible to classify the surgeons. In general, it may be said that the surgeon may be allowed to operate fifteen minutes. After this time either he has finished or has lost his way.

**Early Stage.**—In the very beginning before there are any adhesions the organ of course must be removed. Care must be exercised in ligating the meson. In the highly inflamed state there



may be no free bleeding yet prolonged oozing may follow if proper ligation is not done. The method of treatment of the stump is immaterial. If the cecum is not affected ligation and inversion is preferable. If the cecum is infiltrated and friable inversion should not be attempted, for the stitches but tear out and invite disaster.

**Stage of Adhesions.**—In this stage there is no abscess but the organ is surrounded by adherent omentum or intestinal loops. It is this type in which the inexperienced plays his havoc. His sense of touch does not guide him to the offending organ and an endless amount of traumatism may result before the organ is located. The gut wall may be torn or so injured that infection escapes after the operation is concluded. Experience only brings facility in locating the organ. The operator can save time by locating the ascending colon, making sure of the anterior tenia and persisting in following this until the inflamed organ is reached.

**Stage of Abscess Formation.**—Generally speaking unless the operator is skilled and a walled-off abscess is formed he should desist as soon as the pus pocket is opened. Usually there will be less danger of extending the peritonitis and in the majority of cases the patient will cure himself of his appendicitis. Should the operator persist in finding the appendix the wall of the abscess may be ruptured and the infection be permitted to spread. Sometimes there are multiple abscesses and the drainage of the first may not drain all. This primary drainage may be supplemented by later ones should this become necessary.

**Diffuse Peritonitis.**—When there is diffuse peritonitis and the appendix floats free, it should be ligated and removed. When it is buried in adhesions and the operator requires more than a very few minutes to locate it, he had best allow it to remain and content himself with the simple drainage of the abdomen. The method of drainage is that common to general peritonitis. It need be remarked that the drain, particularly a gauze drain, should *not* be allowed to come in contact with the stump of the appendix, for if it does, the formation of a fecal fistula would be very much encouraged.

The drainage of late abscesses has been covered in the section dealing with retroperitoneal abscesses.

### Prognosis

Nine-tenths of the cases of appendicitis recover from the attack if left untreated. The conflict of therapeutics is waged against the final ten per cent.

**Interval Operations.**—Operations done after the acute attack has subsided should have no mortality. He who operates on a sufficiently great number must likely sooner or later meet a surgical disaster. There are no absolute certainties in surgery.

**Acute Periappendicitis.**—Operations in this stage likewise have a very low mortality, but embolism, secondary abscess, and the like will have a certain mortality—possibly one or two per cent.

**Periappendiceal Abscess.**—With a walled-off abscess the mortality is about the same as in the group just mentioned. Secondary abscesses may form with a prognosis all their own, and must be figured for the individual case. Once a walled-off abscess is allowed to break spontaneously, the prognosis becomes grave, particularly in those younger than puberty and beyond forty years of age. Even secondary abscesses themselves walled off add materially to the gravity of the lesion.

**Spreading Peritonitis.**—When the inflammation is not limited by reactive inflammation, the prognosis depends on the time and character of the treatment. If early drainage is secured with ablation of the infecting area, the mortality should be not over five per cent. If there is notable delay, beyond say twenty-four hours, the mortality mounts rapidly. Attempts have been made to calculate the percentage. It is not possible to calculate the character of the lesion and the degree of violence of the infection, hence a mathematic calculation is not possible. The prognosis of the concrete case must be made on data given in the general chapter on prognosis.

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## CHAPTER XX

### CHOLECYSTITIC PERITONITIS

Inflammation of the gall bladder and the structures lying near it is one of the most common phenomena observed in the abdomen. In the simpler cases the peritoneal covering of the gall bladder becomes hyperemic in sympathy with the circulatory phenomena of the mucosa, less often the peritoneum is actually coactive in the reactive process, without, however, this reaction being sufficiently intense to affect the surrounding organs. Not infrequently the surrounding organs do respond to the gall-bladder involvement, either by simple inflammation or by adhesions limited only to the area in contact with the gall bladder. These are toxic phenomena only. In rarer instances bacteria escape through the wall of the gall bladder and form a more or less spreading inflammation in the peritoneal cavity. More rarely still the gall-bladder wall may become perforated, permitting its contents to escape *en masse* into the peritoneal cavity. This infection may be localized in the region of the gall bladder or it may extend uninterruptedly throughout the abdominal cavity. These various exigencies may be considered *seriatim*.

**Pericholecystitic Hyperemia.**—In most cases of gallstone colic when the organ is observed in the acute stage it shows more or less hyperemia of the peritoneal coat. Even when much reddened and edematous, it may show no real reactive process. This is the state usually observed in the ordinary ephemeral gallstone colic. On section the peritoneal vessels are dilated, here and there a few leucocytes are seen, possibly some ecchymosis and fibrinous exudate, but the state is hardly beyond that of simple hyperemia, altogether analogous to that already described in the general section on hyperemia of the peritoneum. When the source of irritation ceases the hyperemia subsides if not too long continued so that no permanent changes in the vessels take place. When this does occur a varicosity results. This state is more rare over the gall

bladder than over any of the other hollow organs. The surrounding peritoneal surfaces are much more apt to retain evidence of passed irritation than is the peritoneum covering the gall bladder itself. In this respect it is entirely analogous to the conditions existing about the appendix. The cholecystoduodenocolic ligament often shows a permanent hyperemia when the gall bladder itself shows none (Fig. 192). The peritoneum in the region of the colon and beyond likewise may show an increased vascularization. This

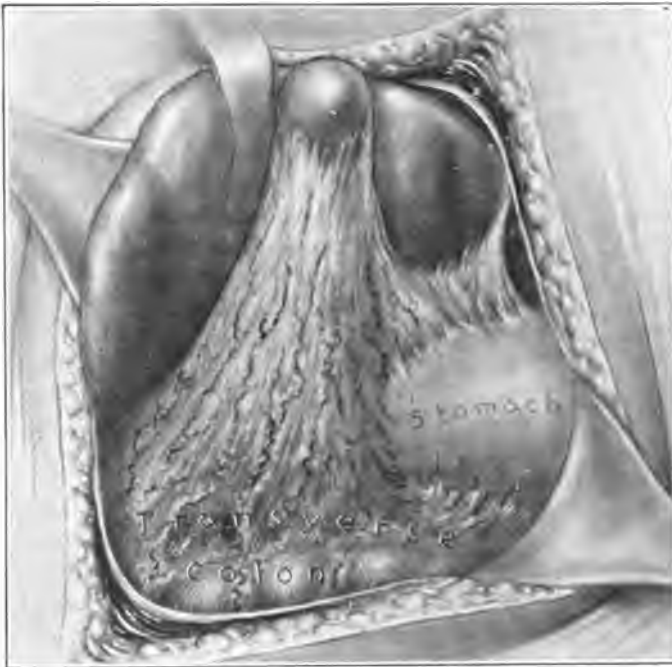


Fig. 192.—Dilatation of the vessels in the hepatocolic ligament in a patient who had had many attacks of cholecystitis, but at the time of operation was free from symptoms.

state of the surrounding peritoneum I believe is a more accurate criterion for the removal of the gall bladder than is the appearance of that organ itself. Like the appendix the wall of the gall bladder may recover so completely that no exact evidence of disease can be pointed out, but it still is subject to recrudescence of the inflammation.

The reaction of the peritoneum covering the gall bladder may give

rise to local tenderness, but does not excite muscular rigidity unless the parietal peritoneum has become excited by contact. Rigidity plus tenderness indicates a wider extent of reaction than tenderness without rigidity.

**Pericholecystitic Peritonitis.**—When the reaction within the gall bladder is more intense, a reactive process on the part of the cover-

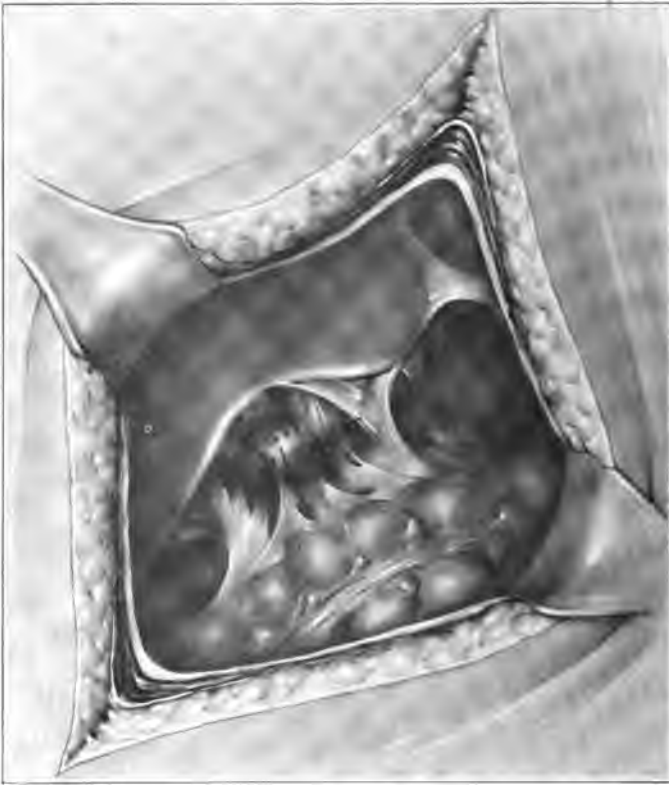


Fig. 193.—Pericholecystitis with adhesions which attach the gall bladder to the colon. The gall bladder was filled with pus and a single large ball-valve stone occupied the beginning of the cystic duct.

ing peritoneum takes place. The vessels dilate, abundant cellular and fibrinous exudation takes place about the vessels and on the surface of the peritoneum. The reaction is usually such that the exudate irritates the peritoneum of the surrounding organs, setting them into a state of hyperemia and corresponding exudation. The

irritation of the surrounding organs is a chemical one. In some instances the fluid may be bile stained, and yet be free from bacteria. The abdominal wall is irritated as well and the recti muscles respond by a defensive rigidity. The result of these opposed exudates is an adhesion of the surrounding organs to the gall bladder. This may be so extensive that when the process is some days old, particularly if previous similar attacks have occurred, the gall bladder may be found with difficulty (Fig. 193).



Fig. 194.—Beginning necrosis of the gall bladder. The outlined areas were made by stones which had lain deeply imbedded in the mucosa. The peritoneum over these regions was blue-black in color.

Usually the exudate on the gall bladder peritoneum is absorbed and the adhesions are released. In exceptional cases the adhesions to the surrounding organs are permanent. In extreme cases the gall bladder may become imbedded in a mass of scar tissue.

**Spreading Peritonitis Going Out from the Nonperforated Gall Bladder.**—In this condition bacteria escape through the wall of the gall bladder because of the loss of its integrity. This occurs in conditions more intense than in the preceding section, yet not



great enough to be attended by actual perforation. In order that bacteria may escape, the wall of the gall bladder must be more or less necrotic (Fig. 194) and the contents must be under increased pressure. This is usually brought about by a stone impacted in the cystic duct or by an inflammatory occlusion of that channel. When this occurs the infection may be localized either by adhesions formed by some previous inflammation or in advance of the actual suppuration in the attack under question. In the absence of this the infection may extend unhindered. When the infection is localized the colon and great omentum usually form the prominent barriers to the advance of the infection. Such infection may go on to abscess formation with its consequences, or it may subside with the subsequent absorption of the barrier adhesions, or they may remain permanently, hieroglyphics of the past catastrophe.

The genesis of these nonperforative pericystitic infections has been the subject of speculation. Schiesselbein and Kehr believe that infection travels by way of Luschka's canals. Clairmont and Haberer were of the same opinion and report a fatal case. Riedel reported three cases, one of which recovered following operation, the others died untreated. Doberauer reported two cases, one of which followed a trauma and the other followed a typhoid ulcer. Hugel reports several cases. This author makes the interesting observation that the reason peritonitis does not more frequently follow cholecystitis as compared to appendicitis is because of the free anastomosis the gall bladder receives from its peritoneal attachment with the liver, in contradistinction to the lack of such anastomosis in the appendix. Johansson reports a case of his own. The changes in the gall bladder were so slight as to give rise to the belief that the perforation may have been in some other part of the biliary apparatus. The author explains its occurrence by extension along the lymphatics. Salager and Roques report a case occurring in childbed. Neuwerck and Lübke doubt if peritonitis can take place without perforation. They conceive it possible that a rupture may have been present but has healed. This hardly seems likely since a gall bladder that is in a state of reaction or degeneration such as these invariably are, would hardly heal during any period of time, much less in the time these cases have been under observation.

This variety of pericholecystitic inflammation is not so rare as the literature would indicate. Pericolic infections of this variety are a common occurrence in the practices of those surgeons who drain acutely inflamed gall bladders. In harmony with the law of the formation of adhesions this type is less likely to be followed by permanent adhesions than the simpler type just discussed. In my earlier work I was often astonished to find at secondary operation made for the purpose of the removal of the gall bladder that all vestige of adhesions had disappeared.

For the most part peritonitides following nonperforative inflammation of the gall bladder are mild in character and tend to regress. Only in a limited number of cases, as indicated by the above citations, do they progress to the death of the patient.

The only bacterial study recorded is that of Hugel. He found coli in several cases, in one streptococci and in one typhoid bacilli. The bacteriology of pericholecystitic inflammations has not been adequately worked out. Gilbert and Lippmann have studied the anaerobes in the normal state. It is possible that these play a role in the pericholecystitic infections. A number of the recorded cases note that the patient had previously had typhoid. Hugel found colon bacilli and typhoid bacilli in several cases and streptococci in one.

**Peritonitis Following Perforation of the Gall Bladder.**—This condition may result without pronounced infection. It is the product of necrotic inflammation plus local pressure. Ulcers similar to peptic ulcers have been reported as resulting in perforation. Frequently perforation takes place over the site of a stone. In these instances reactive phenomena accompany the pressure phenomena. This may result in adhesions to a surrounding organ with perforation into this instead of into the free peritoneal cavity. This is commonly observed in those cases in which huge stones which have so ulcerated through call attention to their migrations by producing an intestinal obstruction. This type of disease is not rare. McWilliams reports on 186 cases. This author records perforation as dependent upon the virulence of the gall-bladder contents and the presence or absence of anticipatory pericholecystitic adhesions. When the escaping material is composed of unchanged or slightly changed bile and mucus, the reaction may not be great.

In such instances the question is not far removed from that of the absorption and local reaction from unchanged bile. Most authors (Naunyn, Thomas) are agreed that no peritonitis is produced, or at most a chemical peritonitis is produced when unchanged bile is absorbed from the peritoneal cavity. My own observations on the effect of bile in the free peritoneal cavity lead me to believe that its presence is more deleterious than these authors indicate. That an animal with a ligated common duct or a patient with an occluded one may live for a long time is no argument for its innocuousness in the peritoneal cavity. By such occlusion the bile is forced into the blood stream via the liver capillaries and may undergo some change, while when absorbed from the peritoneum no such change takes place. At any rate an animal from which the fundus of the gall bladder has been removed dies more quickly than one which has had the common duct ligated. The general opinion is that peritonitis following the rupture of the gall bladder is likely to run a mild course.

When the perforation takes place slowly or previous attacks have produced adhesions, a localization of the process may take place. The process is then like that already described for localized non-perforative peritonitis. Erhardt reported eleven recoveries in thirteen cases. Auvray reported a case dead after eighteen hours. I observed a case in which a woman of twenty-six after being sick with extension to the environment, with the associated rectus rigidity fever up to  $104^{\circ}$ , and a leucocyte count of 30,000, died in collapse within three hours following spontaneous rupture of the gall bladder without the clinical symptoms of a generalized peritonitis. Noetzel and Körte regard gall-bladder perforation as a grave disease. If perforation erodes a large vessel hemorrhage may present an added danger (Graff and Grube). When the contents of the gall bladder is purulent and there are no protecting adhesions the result is a rapidly fatal peritonitis. If adhesions have formed, a local abscess results.

*Symptoms.*—Hyperemia and the milder inflammations hardly distinguish themselves from the symptoms of the gallstone colic. It is only after definite inflammatory changes cause local tenderness, with extension to the environment, with the associated rectus rigidity that symptoms characteristic of peritonitis can be spoken of.

In pericholecystitis with adhesions definite masses may form. The infiltration may be so intense as to simulate malignancy. Diffuse abdominal rigidity with symptoms of sepsis bespeaks perforation. The symptoms of spreading peritonitis then ensue.

*Diagnosis.*—When the symptoms indicate a peritonitis localized in the hepatic region the source can be determined only by the antecedent history. If distinct cholecystitic attacks have preceded, this source of the infection may confidently be diagnosticated. If the gall bladder has become palpable following pain in this region and a peritonitis subsequently ensues, obliterating the palpable gall bladder by the muscular rigidity, the diagnosis is certain. In more obscure cases a diagnosis of peritonitis only can be made and the surgeon must be prepared to find an appendix, lying high beside the colon, awaiting him. Perforating duodenal ulcers are usually attended by more intense pain at the onset with a rapid spreading down the right lateral portion of the peritoneal cavity. Slowly perforating ulcers and sudden bursting of infected gall bladders can be distinguished by the history.

*Prognosis.*—In the simple associated peritonitis the prognosis is altogether that of the disease upon which it is dependent. When an abscess has formed, if well encapsulated and no complications exist, the prognosis is uniformly good if promptly drained. Even when encapsulation seems complete, one can hardly be certain that everything is favorable. Associated lesions or other abscesses may exist. In one of my early patients, operated on by Dr. Dudley P. Allen, death occurred on the tenth day from necrosis of the colon. Neck and others collected 16 cases with a mortality of 33 per cent in the diffuse cases. Körte in his first four cases had a mortality of 75 per cent. Härting reports 27 cases with 15 recoveries. Hirschel recorded 7 cases all fatal. As in other cases of peritonitis, the prognosis is dependent upon the time the disease has existed since perforation when the operation is done. Kehr estimates that nearly all should recover if operated in the first 24 or 48 hours. By the third day the mortality will be fifty per cent, and by the sixth day nearly a hundred per cent.

*Treatment.*—In simple inflammation the treatment is that of the underlying disease. If caused by gallstones they had best be re-

moved. If evidence of pericholecystitic inflammation exists without adhesions the gall bladder had best be removed.

When there are adhesions of the gall bladder with the surrounding organs, drainage without molesting the adhesions will relieve the patient of his suffering, will add little danger of spreading the infection, and will do much toward preventing the formation of a localized abscess.

Kehr believes that it is correct to anticipate perforation in every case of acute cholecystitis by the extirpation of the gall bladder before it perforates. This does very well as a working plan for the past master but for the common man there is room for thought. Personally I prefer to drain and do a removal later unless, of course, the gall bladder is free from adhesions as above indicated. I am afraid to dig a gall bladder out of its adhesions and then remove it. If there is an acute perforation with early signs of generalized peritonitis, operation should be done at once. Drainage as employed in perforated duodenal ulcers is in order.

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## CHAPTER XXI

### GONOCOCCIC PERITONITIS

**Historical.**—Affections of the peritoneum caused by the gonococcus involve chiefly that covering the tubes. The general peritoneum of the pelvis and the peritoneum covering the organs which lie in the pelvis is commonly excited to inflammation when the tubes are infected. The fact that these surfaces lie in contact with the tubes makes it seem likely that the reaction is caused by irritating substances which extend by diffusion rather than by bacterial growth. This reasoning is substantiated by bacteriologic study of the extratubal exudates. Kelly made many investigations calculated to clear up this point and failed to find the gonococcus in the localized inflammation of the peritoneum. My own efforts to demonstrate the coccus in the tubal serosa or subserosa were not successful. When the difficulty of demonstrating the coccus in the tubal mucosa is recalled, such negative studies are not entirely convincing. Some authors deny that a generalized inflammation is possible. Bumm, for instance, did not believe the gonococcus could multiply on a serous surface and McCann believed that gonorrheal infection menaced the peritoneum chiefly because it provided a chance for mixed infection. Wertheim attempted to solve this problem by animal experimentation. By introducing culture media into the peritoneal cavity of animals along with the gonococcic growth he was able regularly to produce a peritonitis.

More recent studies have proved apparently beyond a doubt that while in the very vast majority of cases the process remains localized, in rare instances the gonococcus produces a peritonitis which extends beyond the confines of the pelvis and becomes more or less general. That spreading is most apt to occur at the time of menstruation most surgeons will agree with Charrier. The first to demonstrate the organism in such a case was Wertheim. In such cases he was able to demonstrate the cocci in the endothelial cells but not in the subserous connective tissue. Cushing

reports two cases in one of which the coccus was demonstrated in the smear and in the other by culture. Young succeeded in cultivating the gonococcus from a case. In some cases of more or less diffuse inflammation the gonococcus has not been found even where the clinical evidence, together with the finding of the gonococcus in the vaginal secretion, favored the diagnosis. Northrup reported two such cases and reviews eight cases reported by Comby. Dudgeon and Sargent also examined eight cases. In five, cultures were sterile. In one case diplococci were found which were Gram negative. In one case staphylococci were found, while the others remained sterile. In such instances the coccus evidently has succumbed after producing the reaction.

**Classification.**—From the foregoing it is evident that we can distinguish two types of disease, depending on whether the responsible organism remains within the tube or escapes to the general peritoneal cavity. Since in the first group the site of irritation is within the tube, it may be called a perisalpingitis. The other being a spreading process naturally may be called such.

**Gonorrheal Perisalpingitis.**—By gonorrheal perisalpingitis is meant the reaction imparted to the peritoneum covering the tubes, and, secondary to this, the peritoneum of other viscera coming in contact with them, when the interior of the tube becomes the habitat of virulent gonococci. This term is preferable to the commonly used one of “pelvic peritonitis” because it centralizes the attention on the focus of the disease.

**Pathogenesis.**—The infective agents gaining access to the female genital tract ascend to the mucosa of the tube and there find a suitable environment for their multiplication. This is the primary site of the infection, but does not concern us in this discussion, for the mucosa may be affected without involving the peritoneum. In the general course of events the submucosa and the muscle wall become infected and by extension finally infection reaches the peritoneum. As the infiltration of the tube wall increases, an abundant fibrinous exudate forms extending to the subperitoneal tissue, which results finally in an exudate upon the serous surface. This exudate excites a reaction on the part of the peritoneum covering the tube and in all surfaces which come into contact with it. In some instances fluid bearing gonococci escapes from the free fimbrial end of the tube and in this way infects the



peritoneum directly. In that event the chief site of reaction is about the ovary and a periovaritis is the result. Even in instances where the site of the greatest intensity of the inflammation indicates that infection escaped from the free lumen of the tube, cocci can not be demonstrated in the exudate. Even in such cases the chief site of irritation remains about the tube and it remains essentially a perisalpingitis.

*Pathology.*—The growth of the gonococcus within the tube excites an abundant exudate within the wall of the tube. This exudate is composed of fibrinous material and leucocytes. This begins in the submucosa, extends to the muscle layer and finally reaches the subserosa, and as the peritoneum is approached it responds with a marked dilatation of the vessels, swelling of the endothelium and an abundant exudate upon its surface.

The fluid exudate coagulates into a fibrinoid material and the connective tissue with which it comes in contact swells up and loses its acidophilic properties. In the meshes of this tissue a very abundant leucocytic infiltration occurs. The polynuclears predominate in the early stage while later mononuclears become abundant. The thickness of the tube wall is sometimes enormous, reaching sometimes the thickness of a centimeter or more. This increase in volume is due, chiefly, to the fibrinoid edema and to a lesser degree only to the polynuclear infiltration.

When regression begins the polynuclears decrease and the mononuclears increase in relative proportion. With the decrease of exudate the volume of the tube rapidly lessens. In pure infections the height of the process is reached in 2 or 3 weeks, but when there are associated mixed infections the process may continue much longer and in the case of the streptococcus even for years.

The advent of the peritoneal exudate about a gonorrheal tube is followed by changes in the surrounding peritoneum that run a definite course. This course is parallel with that followed by any fibrin. It is closely simulated by the presence of a blood clot in the pelvic cavity, as is seen in tubal abortion. A disposition to run a course measured by the duration which results from any fibrin irritation, raises the question whether or not the peritoneal irritation is not chemical in nature as Menge thought. As a result of blood studies in pelvic peritonitis from pus tubes and pelvic irri-

tation from the presence of blood clot following tubal abortion, I was struck by the parallelism. It is less intense in the latter condition, but the duration is the same and in character the subperitoneal changes are parallel, though less intense in the case of the blood clot.

In gonorrheal perisalpingitis a fibrinoid exudate forms on the surface of the peritoneum, varying in degree according to the intensity with which the peritoneum reacts. If the irritation is less violent a fibrillar fibrin results which develops into fibrous tissue and the familiar "cob-web" adhesion remains. It is the tube which has been the subject of moderate repeated inflammation which is most apt to present adhesions. If the endosalpingitis is very mild no peritoneal exudate at all is formed and hence no adhesions.

In the more virulent inflammations a large amount of coagulable material is exuded, granular for the most part, but on the surface of which there is a layer of fibrinous material which may result in permanent organization. The granular substratum is absorbed while the surface organizes. There results, then, when the process has been completed a free membrane spreading from tube to ovary or to the gut. The actual thickening of the tubes may be considerable but the bulk of the mass, felt in palpating a pelvis the subject of this disease, is made up of the thickened neighboring organs. The pelvic peritoneum, the small and large guts lying in the pelvis all add to the mass (Fig. 195). The great bulk of the mass is formed by subperitoneal exudations which are capable of complete resorption.

The clinical manifestations of tubal infection by the gonococcus is directly dependent on the degree of irritation produced in the wall and serosa. Pain is produced by distention of the wall of the tube and by irritation of the serosa. The neighboring organs are disturbed in their function both by the irritation of their peritoneal coverings and by the lessened room in which they lie. The exudate, poisoning the general system, gives rise to leucocytosis and fever and the train of general symptoms which commonly attends such disturbances.

*Pain.*—In these cases it is difficult to separate the pain caused by distention from the subperitoneal exudate and that due to irritation of the tubal and particularly of the extratubal peritoneum.

Judging from the relative painlessness of the uterine endometrium when the site of gonococcal infection it is safe to say that the pain experienced is due to the irritation of the peritoneum itself or to the stretching of the tubal nerve plexus. This assumption is substantiated by the vesical and rectal tenesmus so often associated. The character of pain is always that characteristic of peritoneal irritation—sharp and cutting. At first it may be localized in the

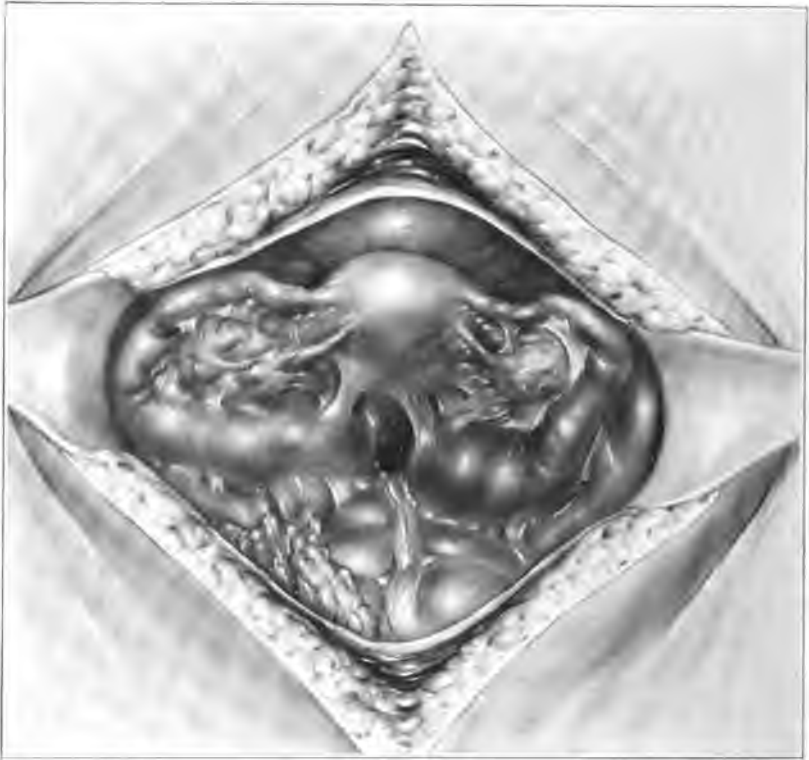


Fig. 195.—Gonorrheal perisalpingitis with adhesions to surrounding organs, subacute stage. As these lesions further regress the adhesion bands lessen and finally "cob-web adhesions" result.

region of the focus of origin, as at the ostium of one of the tubes, and in mild cases it may not extend beyond this point. The initial pain may be so intense as to suggest a tubal abortion. The spontaneous pain in the beginning may be diffused over an area wider than that actually invaded. This is due to reflex diffusion of sen-

sation and to the extensive and extended hyperemia which results from the first irritation of the peritoneum from any cause. Movement imparted by the voluntary acts of the patient, the emptying of the bladder or rectum, because of the involvement of their serosa, tends much to aggravate the pain. These means of aggravation may be imitated by the manipulations of the surgeon either by making pressure over the recti muscles or imparting movements to the uterus or by both as in making a bimanual examination. The pain early in the disease may be so intense as to demand relief. This severe stage usually subsides in two or three days. There remains a sensitiveness to pressure for a week or more.

The diffuse character of the pain is often exaggerated by the patient, or the seat of its greatest intensity falsified. The reason for this deceptive representation may be actuated by reasons of modesty or caution. I have repeatedly seen patients indicate the epigastric region as the seat of the most intense pain. In instances where I feel that the patient is willfully perturbing the scientific accuracy of the clinical study I have made use of a little ruse that has worked successfully in a number of instances. The following is an instance: a young lady had been sick for some days with some acute abdominal lesion the seat of which she declared to be in the epigastric region. Because of the care with which she followed the movements of her environment I suspected she might have been incautious in her social relations. The surroundings were such a high plane of moral purity that I felt I would be taking my life in my hands if any reflecting interrogatory remarks were directed elsewhere than to my cautious inner self. To get a lead I directed the mother that she moisten a mustard plaster and allow daughter to apply it to the seat of the greatest pain and to allow it to remain to the limit of endurance. When I returned the next day the tell-tale patch of hyperemia occupied the space just over the pubis. A look at the patch, a quizzical look at the patient with an apprehensive glance in response, caused me to make a diagnosis of acute gastritis to the mother. The relaxation of relief manifested by the patient at hearing the diagnosis made me sure of the diagnosis of gonorrhea. After recovery the mother was profuse in her thanks for having saved her daughter's life. "Not my

life but my reputation," is what the young lady whispered as she followed her mother from the room.

*Muscular Rigidity.*—Early in the attack the entire abdomen may be distended and the entire wall rigid. This is true even in the absence of diffuse pain. As the disease subsides the rigidity becomes limited to the lower portions of both recti. Rarely, when but one tube is affected, but one muscle may be rigid. Usually then the rigidity is confined to a length of muscle of about three fingers.

*Tumor.*—A mass in the pelvis is the common accompaniment of peritubal affections. Early in the disease there may be but slight thickening of the tube, which may be impalpable because of the rigidity of the muscle above. There is early a sense of resistance due to the edema. This becomes more and more marked until within 48 hours or thereabouts the whole pelvis is as if filled with a plaster cast. A mass appears to bulge in the culdesac and on either side of the uterus. Sometimes only an indefinite resistance is felt by the abdominal hand because of the rectus rigidity. As the mass becomes more dense the sense of tumor is more pronounced, and when the process begins to subside and the muscle rigidity disappears, definite tumor masses are made out so round and distinct that many a surgeon has diagnosticated a myoma. The density of the inflammatory tumor is astonishing and may exceed many of the myomata. It may in fact approach the density of a carcinoma. The great bulk of the tumor is produced not by a free exudate either in the tube or without, but by an extensive edema of the subperitoneal connective tissue. This is especially noteworthy since the beginner is apt to believe that an abscess is present. It is only after the surrounding reaction subsides and the edema and infiltration lessens that the tube itself becomes palpable and pus within the tube becomes of a bulk sufficient to contribute to any noteworthy degree to the size of the tumor. Only rarely does the tubal contents form any considerable proportion of the tumor.

*Temperature.*—The rise of temperature is usually marked—higher than in other diseases producing the same degree of pain. Often 103 degrees is exceeded early in the attack and temperatures of 104 or more are not unusual. After remaining at this height for

several days it begins to subside so that it reaches normal in about ten days to two weeks unless a mixed infection supervenes.

*Blood.*—A moderate leucocytosis is the rule though 20,000 is not infrequently exceeded. I noted one patient with 60,000. After remaining at the initial height for ten days it decreases and reaches normal ten days or more after the temperature has become normal. The leucocyte count reaches normal before there is any considerable lessening of the exudate. The blood count is not a guide to the proper time to operate. A pronounced infection may be stirred up in the presence of a normal white count.

*Diagnosis.*—The diagnosis of this condition may be made simple if a history is obtainable. Those ignorant of their condition will tell the truth as will those who are lost to shame. The class who know their condition and are sensitive to the opinion of society are apt to fabricate and the surgeon must be on his guard lest he misinterpret the information offered.

*Extrauterine Pregnancy.*—When disaster overtakes an extrauterine pregnancy the pain is sudden, but the general reaction is one of collapse—rapid pulse, pallor, low temperature. In gonorrhea with sudden onset of pain these findings are reversed, full pulse and fever. Rigidity and local tenderness are apt to be present in both. Later on the tubal abortion forms a mass, but it is less tender, the temperature seldom is high and the leucocyte count rarely over twelve thousand. The hemoglobin is not altered in perisalpingitis but may be in tubal pregnancy.

*Appendicitis.*—With a low-lying appendix the general symptoms of pelvic inflammation may be present. There may be a history of previous attacks of appendicitis and the rigidity and tenderness is usually marked lateral to the pelvic border. The rectus rigidity is unilateral in appendicitis and a greater stretch of muscle is rigid than in tubal infections. The markedly high temperature may point to a tubal infection. There may be tenderness on vaginal examination in appendicitis, but the cervix is not fixed. I have long studied the state of the pelvic organs in girls when operating for acute appendicitis. I have been struck with the frequency in which one or both tubes show marked reaction, which, had the appendix itself not been inflamed, one might have regarded as primarily an infection of the tubes.

*Ovarian Cyst with Twisted Pedicle.*—When the pedicle of a cyst becomes sufficiently twisted upon itself to disturb the circulation of the sac wall, degeneration begins and the exudate produced excites exactly the same irritating effect on the surrounding tissues that takes place in the gonorrheal tube. Temperature and leucocytosis may be equally marked. On examination a definite tumor may be formed or the patient may have known of the previous existence of a tumor. Nearly always bimanual examination will show a tumor better outlined than a “gonosalpinx” of the same age. In rare instances, a small tumor may be so thoroughly masked by exudate as to be indefinite. In such instances history may be of some use. For instance one of my patients was quite sure the trouble was brought on by lifting a wash boiler. I knew then that it was an infection. A woman with a twisted pedicle never knows what started it. In medicine, as in life in general, persons that are positive either lack discernment, or are bluffing or lying. Of these, the lady with the saxophone tube is the queen.

*Treatment.*—When one allows his mind to travel the road that the treatment of this affection has traveled he sees in his mind’s eye a picture like that from a recent battle field, hilly slope, ever-green, little mounds, eternal granite. Scarcely in any other instance has surgery added so much to the unfavorable prognosis as in the precipitate operating on gonorrheal perisalpingitis. Unoperated on, the disease is rarely fatal; operated on in the acute stage a mortality of about 10 per cent occurs, if one may judge from the literature. Operating after the temperature and leucocytosis subsides is less dangerous, but the fragile tissue makes ideal technic impossible.

Only recently, sad to relate, an eminent surgeon has found that the foci can be handled early by placing in a semi-Mikulicz drain. I have no doubt a sterile brick-bat could be successfully sewed in the abdominal cavity—why one should wish to do either is a difficult question.

The treatment of gonorrheal peritonitis is rest. Packs, hot or cold, sedatives, coal tars, bromides, codeine or morphine may be needed the first few days to secure rest.

After leucocytosis has been normal for a month or two the residue of the disease may be treated surgically if anything remains.

The operation consists of removing the tubes in most cases and releasing adhesions. The removal of both ovaries is never justified.

**Generalized Gonorrheal Peritonitis.**—It is evident from clinical experience that gonococci have no great affinity for serous surfaces, for not alone is the pelvic peritoneum exposed early in the disease by way of the tubal ostia but frequently in the course of operation pus containing cocci has escaped without producing a spreading.

*Pathogenesis.*—The greatest care is required in the interpretation of spreading infections following gonorrheal endosalpingitis. Thus Menge in eight cases found gonococci in but three of them. Even in one of these which ended fatally, streptococci were found within one hour after death. On the other hand the failure to demonstrate cocci in a smear may not be sufficient evidence to exclude a gonococcus infection. For instance in Cushing's first case cocci were obtained neither by smear nor culture. Kiefer in the rupture of forty gonorrheal tubes saw no spreading of the gonorrheal process and concludes that extension is not possible. Yet very often after operations on acutely inflamed pus tubes a generalized peritoneal irritation results, whether due to irritation or from actual growth of bacteria is difficult to say.

My remarks relative to the pathogenesis of gonorrheal perisalpingitis were based on researches repeating the experiments of Wertheim. My method was to implant a pus-soaked bit of gauze into the peritoneal cavity, thus at once producing the irritation by a foreign body and providing the organism with a favorable environment for its development. Wertheim injected cultures into the peritoneal cavity of animals and observed a nonfatal peritonitis which reached its height on the third day. He found that the cocci possessed the faculty of penetrating the endothelium for he found them in the muscular layer of the guts. My studies indicated that there was an early development of the gonococci. That the cocci should develop after the introduction of a foreign body is not surprising, for when the foreign body is introduced a serous exudate forms about it producing a culture media of the proper temperature, rivaling in perfection of appointment the most painstaking imitations in the test tube. The fact that these cultures invariably die within 72 hours shows how inimical is the environ-



ment. Nicolaysen found that mice died after injection without the production of a peritonitis. This result likewise followed the use of sterilized cultures.

The escape of pus from the end of the tube provides an irritant which ordinarily acts only on the adjacent serous surfaces. Possibly the infection does not always travel by way of the tube. Goodman reports one case in which the generalized peritonitis developed on the third day after confinement and he concludes that the infection traveled by way of the lymphatics. The frequency with which a pyosalpinx begins at the time of menstruation makes it seem possible that the culture media furnished by the lochia may have aided in the development of the gonococci. At any rate a lymphatic transmission can not be proved. In most of the cases reported the tube showed involvement or with care pus could be pressed out of a tubal ostium.

Generalized infections are possible as the endocardial affections due to the gonococcus demonstrate. In a case of general peritonitis Colombini found vegetations on the valves of the heart. In one of Hunner and Harris' cases a diplococcus was recovered from the blood stream. Metastatic septic emboli developed in Frank's cases.

Why in rare instances the tubal infection extends beyond the confines of the pelvis and reaches the ultimate region of the abdominal cavity is not known. In some instances of generalized abdominal inflammation careful search by competent men has failed to demonstrate the gonococcus further than the lumen of the tube. In other instances of like clinical pictures cocci are demonstrated both in cover-slip preparations and in cultures from the remote regions of the abdomen. The parallel clinical pictures warrant us in assuming that those cases which were bacteria-free were due to the same bacterial cause. In some instances clinicians have no doubt gone too far in arguing from the minor premise and have diagnosticated a diffuse gonococcal peritonitis when there was no other evidence than the coincidence of a vulvovaginal Neisserian infection and a generalized peritonitis. I once saw a perforation at the base of the appendix in a girl who had recently acquired a gonorrheal infection.

*Pathology.*—The exudate in diffuse gonorrheal peritonitis is

small in amount, of a greenish color and usually clouded by flocculi of fibrin. The intestinal coils are sometimes agglutinated by the fibrinous exudate. The affection is often diffusely distributed and the region about the liver is particularly involved. The subperitoneal involvement is apparently not great. There is no evidence available bearing on the question of adhesions in the diffuse variety. The pathology is wholly different than when the disease is limited to the pelvis.

*Frequency.*—Diffuse gonorrheal peritonitis is one of the rarer diseases of the abdomen. It is well to follow the plan of Hunner and Harris who divided their cases into those in which there was bacteriologic proof and those in which the diagnosis was made on general grounds. Of the former class they were able to present fifteen from the literature and added four more. Of the latter, they gathered sixteen from the literature and added three more.

Since this paper a number have been reported. Goodman adds three cases, all diagnosed on clinical grounds.

*Age and Sex.*—The majority of the cases, as would be expected, occur in adult females. Seven, however, were girls under fifteen years of age. A few have been reported in males. Information relative to the conditions in the male is still more unsatisfactory. V. Zeissl reports three cases. He, himself, expresses skepticism whether or not the gonococci played any role in the peritoneal process. In each of his cases there was an epididymitis and all recovered. Mermet reports a more plausible case in which there was vomiting and a temperature of 40° C., painful abdomen. The symptoms in this case seem to have paralleled the testicular improvement. In a similar case in my experience the abdominal symptoms disappeared within a few hours after the epididymis was drained. I have several times noted that recurrent acute abdominal symptoms coincided with recurrent epididymitis both of which ceased to recur simultaneously with the relief of the latter affection. The pain is due to inflammation of lymph glands along the course of the vas. I doubt very much if a real inflammation of the peritoneum due to the gonococcus has occurred in the male.

*Symptoms.*—The onset may be relatively slow but usually it is stormy. Chill, abdominal pain, vomiting, distention marked the beginning in a number of the recorded cases.

In some instances pelvic infection preceded the more violent abdominal symptoms, but in the majority the severe symptoms were the initial complaints. In many there was a preceding vulvovaginitis. The characteristic feature, according to Hunner and Harris, is the sudden improvement after a few days of a stormy course.

The temperature is only moderate, varying from  $100^{\circ}$  to  $102.5^{\circ}$ . In some instances it has been very high, even to  $105^{\circ}$  and over. On the whole the temperature tends to range higher than in pus-microbial peritonitis producing like symptoms.

Leucocytosis is usually moderate, varying from 15,000 to 20,000, though Goodman reports one case with a leucocyte count of 60,000. The polynuclears range low, from 60 to 80 per cent.

*Diagnosis.*—The stormy onset may detract the attention from the seat of its origin. Hunner and Harris believe that the surgeon should be so familiar with the picture of peritonitis arising from other sources that the symptomatology of gonococcal peritonitis should excite his curiosity. The demonstration of a recent or more remote genital infection makes such a diagnosis probable if the history indicates the origin of the pain in the pelvis. This probability is much heightened by the discovery of salpingitis or perisalpingitis. If the general symptoms follow manipulation of the tubes or during the menstrual period or puerperium with the known presence of gonorrheal infection the diagnosis may be made almost with certainty. There is little to add to the opinion of these observers.

If the abdomen is opened before the diagnosis is made, the acute dry plastic peritonitis may give the operator his clue and excite him to the proper investigations necessary to make a diagnosis certain.

*Prognosis.*—In the 39 cases reported on by Hunner and Harris, of the 24 cases operated on, 19 recovered and 5 died, of the 15 not operated on 8 recovered and 7 died.

*Treatment.*—In some of the cases operation was done before a diagnosis had been made. During the period when it was considered proper to operate pus tubes in the acute stage naturally a generalized gonococcal peritonitis was regarded as a fit object of attack. With the reaction against operating acutely inflamed

tubes, doubt as to the correctness of operating on the generalized type likewise arose. Most of the more recently recorded cases have been managed by masterful inactivity.

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## CHAPTER XXII

### PNEUMOCOCCIC PERITONITIS

A general infection of the peritoneal cavity by the pneumococcus is not a frequent malady if the paucity of reported cases be accepted as a criterion. It is worthy of note that those who have studied this type of peritonitis and have learned to recognize it usually have several cases to report. From this one is led to suspect that the majority of cases are overlooked. Aside from its clinical importance it possesses an interest because of the fact that it sheds some light on the genesis of cryptogenetic peritonitides in general. Our knowledge of this malady is dependent very largely on studies made by the French. English and particularly American literature has contributed but very little, aside from a few case reports. The reason for the preponderance of the information available from continual sources is not clear. Climatic or racial conditions may be a factor which makes the disease more frequent in continental Europe, but one can hardly repress the fear that so little has found expression in English because minds employing this language for the expression of their ideas have not given themselves to the necessary intensive study of the problem.

**Historical.**—Broussais records several cases of acute idiopathic peritonitis which he regards as rheumatic which probably belong to this class. Duparque under the head of "essential peritonitis in young girls" presents the first complete account. Féréol emphasizes the disposition of this type to terminate in spontaneous perforation at the umbilicus. Gauderon essays a collective report and analyzes 25 cases.

The modern history begins with Bozzolo. As the title of his paper indicates (*Ueber eine Form durch Kapselkoken verursachter multipler Serositis, eingeleitet durch Erscheinungen eines akuten morbus Brightii*) he was able by virtue of the then recent bacteriologic investigations of Fränkel and Weichselbaum, to identify the specific organism. Complete recent articles have appeared in the

German literature. V. Brunn reports 2 cases and collects 72 from the literature. Rohr reports 9 cases and gives a complete review of the literature.

**Pathogenesis.**—Brunn was able to produce a localized peritonitis by injecting pneumococcic sputum into the peritoneal cavity. Boulay produced localized peritonitis by injecting some insoluble substance like gelatin with the pneumococci. Jensen succeeded in producing a purulent peritonitis by the simple injection of the pneumococcus into the peritoneal cavity. I was able to produce a local peritonitis only by implanting a pledget of cotton soaked in a bouillon culture of the pneumococcus. Cultures injected directly into the peritoneal cavity either produced no marked effect or killed the animal, apparently from toxic absorption.

As above noted the chief interest lies in the question as to the possibility of hematogenous infection. Boulay by first introducing a foreign body into the peritoneal cavity was able to produce a peritonitis by injecting the pneumococci subcutaneously. My own attempt in this direction resulted in death by septicemia with the usual inflammatory reactions about the foreign body.

In examining such a local irritation for bacteria in an animal dead of general septicemia great care is required not to contaminate the local field from the blood stream. This can best be avoided by washing the animal's circulation free from blood with formalin solution and then searching for cocci by tissue staining methods. Desguin assumes the extreme position that all pneumonias are primarily septicemias with later localization in special regions.

Opposed to this theory is that of Burekhardt who believes that there is a direct transmission through the diaphragm. Bozzolo demonstrated a transmission in 15 cases of pneumococcic pleurisy. A possibility of the reverse must be kept in mind. Krogus reported cases in which pneumonia followed a general peritonitis. This possibility is attested to by our everyday experience of lung complications from the pus organism arising in the abdomen. In order to establish the development of a pneumonia secondary to a pneumococcic peritonitis it is necessary to demonstrate a local origin for the primary pneumonic process in the peritoneum. Pus organisms metastatic in the lung may, by producing a reaction in the lung, stimulate the ubiquitous pneumococcus to development. A

frank pneumonia complicating a peritonitis is no evidence of the pneumococcic nature of the peritonitis, nor even that the lung affection is pneumococcic in origin. This is evidenced from the fact that in such cases abscess frequently results from which pus cocci may be recovered, indicating that the whole disease was due to pus organisms.

The researches of Jensen sought to establish a hematogenous pneumococcic peritonitis via the intestinal tract. He fed young rabbits, without previous intestinal lesions, virulent bouillon cultures of streptococci and secured a peritonitis and Geirswald got the same result with pneumococci grown in milk. Jensen found necroses in Peyer's patches. The source of the infection from the mouth via the intestinal tract is easily hypothecated, but satisfactory proof of its occurrence has not been provided. The fact that the pneumococcus of the healthy sputum readily produces septicemia in animals can hardly be entered as an argument. Dieulafoy, discussing possible avenues of entrance, argued for the stomach and Weichselbaum for the intestines. Flexner reported two cases in which extension from the gut tract is likely. Lennander and Nyström report observations in the human subject in which there was swelling and reddening of the gut wall with an associated peritonitis. By microscopic examination they were able to trace the infection directly through the wall of the intestine. Stoos reports a case in a girl of five who died on the second day of the disease. The solitary follicles and Peyer's patches were swollen and the superficial surface necrotic. Microscopic examination showed diplococci in all of the layers of the gut. Peritonitis going out from an infection of the appendix from which the pneumococcus alone was recovered, was reported by Canon, and a number of others. A diplococcus has been recovered in many cases of appendiceal peritonitis. For instance Krogius in forty cases found such an organism in twenty-one of them. Neither the identity nor the etiology relationship of these organisms was positively established. There seems to be but little doubt but that such a route may be followed by the infection, but there is no evidence that the gut wall is more pervious to pneumococci than to other bacteria.

As yet no case has been proved to be of hematogenous origin. Zesas makes the statement that experimentally no case of peri-

toneal infection has ever been produced by injecting organisms into the blood stream. On the other hand Michaut believes that the blood stream is the only avenue of infection and v. Brunn regards this as the most obvious channel. Desguin regards peritonitis as merely a local expression of a septicemia. Cuff comes to a similar conclusion. The existence of the disease in the meninges and peritoneum in an infant three days old leads Netter to assume a placental infection. A similar case is reported by Czemetshka. Finally Rohr argues from analogy that since pneumococcic abscesses in the thyroid, in the joints, and in the medulla of bone occur, it must be assumed that the infection gains entrance through the blood stream.

Because the lungs are the natural field for the exercise of the activities of the pneumococcus it is natural to suppose that the primary lesion should be sought there. The hypothecated direct communication between the two great serous cavities would seem to increase the probability. Notwithstanding these *a priori* arguments, clinical experience does not seem to bear out this assumption. Rohr in his nine cases found nothing more than a simple cough preceding the peritonitis in two cases and no pulmonary symptoms in the remainder. V. Brunn and Jensen regarded the concurrence as rare. However, there are a number of instances where a frank pneumonia seems to have preceded the peritonitis. Burckhardt has reported such cases, as has Mathews. Bowen, Anand, and a number of others report cases equally as convincing.

Nevertheless, as a complication of pneumonia, peritonitis is not a very frequent disease. Fawcett in 182 autopsies on bodies dead of pneumonia noted that in only 5 was there an involvement of the peritoneum. In this country Pearce found the association only 4 times in 121 bodies.

Because of the preponderance of females affected, at least in children, it is quite natural that the genital portal should be interrogated. Dudgeon and Sargent have reported a case of diplococcic peritonitis in which a pyometrium was the most pronounced lesion. Notwithstanding the eminent qualifications of these investigators one can not help recalling that though the patient was only eight years old there was an associated gonococcal infection.



Riedel reported a number of cases associated with inflamed tubes from which pus could be squeezed.

In adults, likewise, a number of cases have been reported in which pneumococcic peritonitis was associated with salpingitis. Pearce reports one in which a pneumococcic pyosalpinx ruptured causing a peritonitis and two in which the pneumococcus could be demonstrated in the endometrium. Jensen reports a similar case. Canon saw a pneumococcic peritonitis associated with a carcinomatous uterus. Meyer saw a case arising in the puerperium and Pearce reports a similar case. Patellani Rosa found pneumococcic salpingitis 13 times in 945 cases of tubal disease. Since Netter was able to demonstrate pneumococci on cover slips in many cases of pneumonia, v. Brunn's suggestion that since there never has been reported a case of localized pelvic peritonitis due to the pneumococcus, tubal disease due to this organism may be the result of bacteria, at first free in the peritoneal cavity, gaining entrance from the fimbriated end, deserves consideration.

A number of rarer foci have been reported. Netter believes the middle ear is a frequent portal of entry in children. Stone reports a case in which chronic otitis media followed a pneumonia and after several years a peritonitis developed. Canon reports a case developing from a pneumococcic infection of the gall bladder. V. Brunn reports two such cases. Bastianelli reports a case going out from the urinary bladder.

**Pathologic Anatomy.**—The pus is thick, creamy in consistency and of a greenish yellow color and usually odorless. Sometimes there is an admixture of blood. The exudate is usually abundant. In conformity with the usual action in the lung the production of fibrin is abundant. Because of the abundant fibrinous exudate adhesions are abundant and early. It is due to this abundant adhesion formation that the disease is often localized. Once localized the rapidly increasing exudate pushes the unaffected area to one side. In this wise the fact may be explained that even in cases with enormous abscesses the greater area of peritoneal surface remains unaffected. When walling off does not take place the individual coils of gut are agglutinated so that the entire packet may be lifted up together.

The usual site of the abscess is below the umbilicus. This ac-

counts for the disposition of the navel to protrude as soon as the accumulation has assumed large proportions.

Secondary or at least concomitant affections are often noted. The lungs and middle ear are most often affected. These associated lesions are regarded as etiologic factors by some writers and as complications by others. Perhaps each view represents the truth in individual instances. Abscesses in the kidneys and liver may with better reason be regarded as complications.

In adults the pathologic anatomy is less typical. The fibrinous exudate is less in amount and consequently the tendency to adhesion formation is less marked.

**Frequency.**—The literature in general warrants the general statement that the peritoneum is involved in one per cent or less of all pneumonias. Netter found 2 cases in 140 cases of pneumonia. Jensen was able to collect 86 cases out of the literature and to these he was able to add 20 cases of his own. Since that time several small groups of cases have been recorded, numbering in all now about 160 cases. Many of these cases are not established beyond a doubt. The finding of a diplococcus hardly rises to the dignity of a scientific proof of fact. Among those cited by American authors two were by Flexner, six by Pearce, one by Stone, three by Woolsey, and five by Mathews. This seems to represent about all the authentic cases published in this country.

Children are most frequently affected and among these girls are most often affected. Thus in 52 cases under the age of 15 years, 45 were in females. In adults the sexes suffer in about equal proportion.

**Symptoms.**—The pneumococcus being like the gonococcus, capable of producing abundant fibrinous exudate, we may anticipate that this form also will be characterized by a tendency to localization. Our expectations are only partly realized for many of these cases become diffuse quickly. In the main considering that there is, so far as we know, no preliminary reaction on the part of the peritoneum the course of the disease is relatively mild.

The disease, in conformity to custom, may be discussed under the heading of localized and generalized forms.

**Localized.**—There is usually a history of sudden onset of pain in the abdomen, resembling in this regard a perforation. The pa-

tients in many cases state the precise moment of the beginning of the disease as they do in perforation. The pain is usually intense and may be at first generalized. Vomiting seems to be more constant than in many other forms of peritonitis. It is nearly always present at an early period of the disease and usually lasts some days. Diarrhea is emphasized by many authors as being a characteristic feature, notably Stoos, Lenormant and Lecène, and Hawkins, while Rohr found this symptom in 4 out of 9 cases only. Temperature is usually high though there have been marked exceptions, as noted particularly by Annand and Bowen. Most of the authors compare the temperature curve with that of croupous pneumonia which is in a measure justified by the sudden drop observed in some cases. The possibility of different modes of onset may account for this discrepancy. A primary bacteremia may be associated with the sudden rise while the lower temperatures may be associated with a more localized conflict. Herpes has been noted with considerable frequency and its importance is adjusted highly by de Quervain. Koós noted one case in which the lesion was suppurative. Convulsions may occur in young children and chill is a common preliminary symptom in older ones. Sleeplessness and headache may be early symptoms. The headache may be so intense as to suggest meningitis.

As occasional symptoms, epistaxis, vesical tenesmus, jaundice, albuminuria and the diazo reaction may be mentioned.

There is usually a leucocytosis, there being an increase particularly in the large polynuclear variety. In these, according to Haim, there is a very marked fibrinous network and v. Brunn noted an increase in the glycogen content.

The general course of the disease is milder than other varieties of the disease of apparent equal initial intensity. Though the pulse may be rapid and the abdomen distended, the general expression is not that of a severe peritonitis. Muscular rigidity is less marked, despite the intense pain, and it tends to subside earlier. The temperature usually subsides gradually but with greater rapidity than in pus microbe peritonitis and may according to Comby and Grancher end by crisis.

The exudate usually forms in the lower abdomen or near the umbilicus projecting more or less to one side, but may extend up-

ward even to the diaphragm. The site of the abscess is sometimes marked by edema of the abdominal wall or a dilatation of the cutaneous veins. The exudate may be more evident from percussion than by palpation since there is but little exudate in the surrounding tissue. The abdominal wall may be soft and a little tender over the site of the abscess, in fact the presence of abscess has been repeatedly overlooked until threatening perforation or diagnostic aspiration has revealed its presence. When there is greater induration the pyocyanus may be associated, according to Desguin. Because of the flaccidity of the abdominal walls fluctuation may be made out. Sometimes this is distinct and v. Brunn and Sevestre have resorted to exploratory puncture even early in the disease.

If the abscess is not drained the general condition becomes reduced, fever ascends, the abdomen becomes distended and according to v. Brunn takes on the appearance of tuberculosis.

If relief is not provided the umbilicus may bulge, become edematous, then reddened and finally perforate. Rohr regards this as one of the most characteristic features of pneumococcic peritonitis. It is worthy of note, however, that no more than five cases have actually come to perforation.

While pneumococcic peritonitis is in general less stormy than other varieties the likelihood of spontaneous regression is less, though not impossible, even without spontaneous rupture. Such cases have been reported by Broca and Brown.

Rupture in other regions as in a hernia, in Scarpa's triangle, and the bladder have been noted. Rupture into the gut is rare.

**Diffuse.**—The diffuse variety may be regarded as typical for the adult as the localized is for the child. It varies but little from that of any other etiology. Chill, high fever, intense pain are less usual introductory symptoms than in the circumscribed forms. Early tympany is the rule, though the abdomen may remain flat throughout the attack. The rigidity of the abdominal wall is usually relatively slightly marked. The course may proceed even more violently and run its course in a few days under the picture of a septicoptemia as recorded by Schabad and Burekhardt. Even more violent was the course of the disease in a patient reported by Mace, in whom death occurred with the picture of a perforative peritonitis, yet the autopsy by Ophuls revealed no perforation but a pure culture of pneumococci.

**Predisposing Causes.**—That the time of the year should exert an influence on the frequency of pneumococcic peritonitis is not surprising when the greater frequency of lung affections during the winter months is remembered. General debilitating diseases such as tuberculosis, cirrhosis of the liver, malignant growths and the like also seem to invite the disease. Trauma to the abdomen has been noted in two cases of circumscribed peritonitis, one by Dieulafoy and one by Galliard, and two of diffuse, one by Hagenbach-Burchardt and one by Michaut.

Preliminary or associated pulmonary affections, such as bronchitis, pneumonia, bronchopneumonia, pleurisy and empyema are the most common associated lesions.

A variety of commonly associated lesions has been noted. It is often difficult to determine whether these exist as predisposing factors, concomitant infections, or complications. Aside from the lung conditions above noted may be mentioned meningitis, endocarditis, diseases of the middle ear, the parotid, thyroid, testicle, and bone marrow.

**Diagnosis.**—The paucity of literature in English-speaking countries may in part at least be ascribed to the fact that associated lesions frequently dominate the picture, and the peritoneal factor goes unsuspected. The inherent difficulty in diagnosis aside from the associated lesion has frequently been emphasized. Duckworth and Marsh, and Bowen believe the diagnosis is not possible without incision. Dieulafoy on the contrary finds the clinical picture very characteristic.

Because of the specific nature of the affection the demonstration of the pneumococcus must be considered essential to the establishment of the diagnosis. Such a demonstration demands a high degree of skill of the observer in bacteriologic investigation. The morphologic, tinctorial, cultural and toxic characteristics all must harmonize before a positive diagnosis is warranted. Because of the faint cultural vitality of the organism even when present it can not always be identified by this means, as has been emphasized by Krogus. The morphologic identification is sometimes made difficult because of the fact that frequently they do not retain the stain by Gram's method, as has been emphasized also by Krogus, as well as Jensen. Still more confusing is the fact that in old abscesses the morphology is modified. (Bozoolo, Bryant.) These dif-

difficulties are multiplied when other organisms, particularly the colon bacillus, are present. When all these difficulties are taken into account, it is easily understood that while the demonstration of the cocci is the only scientific means of diagnosis, its recognition is often fraught with difficulties.

In order to supplement the bacteriologic examination of pus the blood has been studied by a number of investigators. Jensen, because pneumococcus can be recovered from the blood stream after the injection of cultures into the peritoneal cavity, enthusiastically recommended this means as a diagnostic measure in peritonitis and reports two cases in which the results were positive. Canon has found this method of little use and explains the failure to recover the cocci to the fact that the diseased peritoneum does not absorb organisms as does the normal.

Because of the difficulty of bacterial demonstration diagnosis by exclusion must receive more than ordinary consideration. The usual varieties may be considered *seratim*. Unfortunately there is but little of more than uncertain value.

*Appendicitis*.—Rohr notes that diarrhea, because of the frequently associated enteritis, is more apt to be present than in other forms. This may be of some service in adults but does not obtain in children, as pointed out by Jensen. The absence of a history of previous attacks has been advanced as of positive value, but the literature does not bear this out, notably the statistics of de Quervain and Rohr. The greater frequency of pneumococcic peritonitis in girls and the pus varieties in boys is likewise quite useless when confronted by a concrete case. The difficulty finally is increased by the cases of typical appendicitis due to the pneumococcus.

*Tuberculous Peritonitis*.—Late in the disease the disposition of tuberculous peritonitis to produce a bulging of the navel may simulate this same characteristic of pneumococcic peritonitis. The history of the diplococcic variety is usually more acute, though the disposition of the tuberculous affection is often to develop suddenly after some bronchial affection but it is particularly to be noted that these usually proceed without pain.

*Gonococcic Peritonitis*.—Broca states that a differentiation between the diplococcic types can not be made early in the disease. The same stormy beginning is present in both, vomiting, severe pain, and often diarrhea. Dudgeon and Sargent note that meteor-

ism is more common in the pneumococcic variety. Rohr notes that herpes of the lips points to the pneumococcic variety while vulvovaginitis points to the Neisserian type. When a pneumococcic peritonitis is associated with a gonococcic vulvovaginitis, as has been reported by Dudgeon and Sargent, and others, it is a problem for the courageous bacteriologist.

*Typhoid Peritonitis.*—Waldo reported a case in which there was roseola in pneumococcic peritonitis. The slow beginning of typhoid and the increased leucocytosis in pneumonia usually suffice to make the differentiation. Peritonitis in typhoid usually appears in the second half after the diagnosis has been established. That it is possible to become confused I know from experience. A boy of eleven became quite sick with a temperature of 104°. There were subcrepitant rales over the left lower lobe. On the second day marked abdominal pains with vomiting and tympany set in. Despite this beginning he settled down to a regular typhoid course. It is worthy of note that he had a leucopenia from the beginning.

The essentials for making a diagnosis are obviously that the surgeon thinks of the possibility of pneumococcic peritonitis and that the necessary means of investigation be at hand to prove the point. The rarity of this combination is sufficient to account for the paucity of American literature on this subject.

**Treatment.**—Because of the uncertainty of the clinical diagnosis the deliberate planning of a line of treatment based on theoretic grounds is of little value. Usually the surgeon will discover only after the operation that he has been confronted by a pneumococcic peritonitis. When presumptive diagnosis has been made from the character of onset or by puncture of an abscess, or perchance from a blood culture a planned operation may be possible.

It goes without saying that some have advised immediate operation, some delay—a discussion would not be orthodox that did not recognize these two groups.

If there is a localized abscess, operation is indicated. All can agree on that point but that one should wait until fluctuation can be demonstrated as advocated by Broca will hardly be assented to by many. In the diffuse type an expectant treatment likely would be advisable, but most operators likely will operate under the general diagnosis of peritonitis and will proceed as is their habit in this affection. Fortunately this is a problem few of us

need worry about, for should we encounter such a condition it will cause us no chagrin for we should never know it.

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## CHAPTER XXIII

### PUERPERAL PERITONITIS

The earliest accounts of diffuse peritonitis in the literature have to do with those arising during the puerperium. The student who wants to discover a real thrill in medical literature can obtain it by reading the old accounts of childbed fever. Poe's most weird tales are tame in comparison. These accounts are not without their humorous aspects. The explanation the old observers gave for the presence of the pus in the belly rivals some of the clinical deductions of our own times. As an example it may be mentioned that one theory was that the pus in the abdomen was milk due to the exudation of this fluid into the abdomen rather than escape through the breasts. To prove this casein was demonstrated and one enthusiastic writer even churned butter from fluid obtained from the peritoneal cavity.

Should a student find himself lacking in respect for his profession he can renew his faith by acquiring a knowledge of the discovery of the cause and the elimination of what to me is the most tragic disease to which flesh is heir. It is well to remember that the infectious character of puerperal fever was deduced by the observation of epidemics. The first to formulate the infectious theory was our own O. W. Holmes. Its elaboration and proof by the ill-fated Semmelweis furnishes one of the most heroic and inspiring accounts of achievements by medical men. That infection came from without was definitely established and only later developments were required to show what it was that came from without.

It was my uncanny privilege to locate in a rural community neighbor to an old school (or schoolless) practitioner who never washed his hands before making digital examinations and but seldom afterwards. Death and disaster followed his trail and now more than a score of years after I can not contemplate the experience without a shudder. Through his ministrations I was thus enabled to see all too many patients presenting the clinical signs about to be described. I want to say that no matter how many

patients an observer may have observed suffering from perforative peritonitis legitimate to the present day, the awfulness of the disease can not be appreciated unless he has observed the diffuse type that occurs in the recently delivered woman.

I can not refrain from recording here my first experience with this disease. A woman in her eighth puerperium had been overcome on the third day after labor by a violent chill and high fever. When I saw her on the fifth day she lay motionless, eyes sunken, wide open, and fixed. Her respiration was labored and rapid and despite this labor her color presented a mixture of waxy pallor and cyanosis, as though some vulgar hand had soiled a marble statue of Distress, or Nature herself was seeking to soften the awful picture to spare the untried sensibilities of the embryo Aesculapian. The distended intestine found little resistance from the lax abdominal muscles and ballooned out to an astonishing degree. My first thought as I saw the patient lying in bed was that a canopy had been formed for her out of barrel hoops to prevent friction from the bedclothes. My astonishment at finding that the whole mass was belly knew no bounds. My eyes at this sight I am sure rivaled the patient's in fixity and wideness and my respiration was equally labored. As I sought to feel her pulse the cold clammy skin made me shrink and as I sought the pulse I could find but a quivering string and because of the pounding of my own heart I never knew its rate. As I turned from this scene, standing about the room were the seven older children, the eldest a girl of twelve. These, too, were wild eyed and short of breath. Approaching the cradle I sought to calm myself by viewing the child. Much to my consternation here lay a replica of the mother herself. The infant vainly sought to emulate its mother in girth of abdomen but far exceeded her in rate of respiration. In one particular only was there essential difference. Instead of the waxy gray of the mother it presented a peculiar ochre yellow, the result of cord infection.

In all that scene there was but one calm face—that of the family doctor. Seeing my discomfiture he chuckled derisively and remarked, "Never saw anything like that, did you, boy?" Glancing at his dirty breeches which long had done duty as a handkerchief through an attack of coryza, and at the dirtier hands which because of the inefficiency of their natural cleaner had lost most of

their flexibility, I replied, "No, you dirty old devil, and I swear before the shades of the lamented Jaggard that if scrubbing my hands will prevent it, I shall never gaze on such a scene of my own making."

**Etiology.**—More than a century ago Gordon noted that puerperal fever was due to an infection since it "seized such women only as were visited or delivered by a practitioner, or taken care of by a nurse who had previously attended patients affected with the disease."

We know now that this infective agent is some sort of organism. The streptococcus is the organism most often present. MacDonald estimated that this is the causative organism in 40 per cent of the cases. Lloyd gives the statistics in 159 cases. Streptococci were found in 35 cases, staphylococci in 30 cases, pneumococci in 17 cases, gonococci in 21 cases, colon bacilli in 22 cases and the bacillus aerogenes capsulatus in 2 cases. The course of puerperal peritonitis strongly suggests the presence of the streptococcus, and when the ubiquitous staphylococcus or colon bacillus is present the identification of the streptococcus is a very difficult matter. Even when present the pneumococcus or gonococcus can with difficulty be conceived as the sole or even chief offending organism. Confronted by such a stormy disease as puerperal peritonitis it is as difficult to establish an alibi for the streptococcus as for a small boy when confronted with an empty jam jar and a jam-besmeared face. Sister may be the culprit but there is the prejudice born of experience.

The question as to the source of the infection can be discussed in polite society only in the abstract. It usually comes from without and since the attendant is usually an inhabitant of that indefinite space called the environment of the patient the burden of disproof rests on him. When confronted with a concrete case it is well to remember that the infection may be supplied by the patient herself. The patient may have a pneumonia as recorded by Baisch. A suppurating myoma has been a cause, as reported by Lepage and Mouchotte, and from suppurating cysts as recorded by Patton. An appendicitis may produce the infection as emphasized by Findley. It is evident from these few citations that puerperal peritonitis may be caused from some lesion within the patient herself. At

the same time when a practitioner has the misfortune to have this disease develop in a patient of his the verdict should be that rendered by a trial board against a too amorous minister, "Not guilty, but don't let it happen again."

**Pathogenesis.**—Bacteria gaining entrance through the genital tract by the introduction of some unclean thing presents the commoner mode of introduction of the infection. Peritonitis, the problem which concerns us here, represents but one of the possible disease entities which might result. Metritis, pelvic cellulitis, septic thrombosis local and at a distance, and pure septicemia are but some of the possibilities, aside from the infection of the peritoneum, that may occur in the puerperal period. No student of genital infections may consider himself possessed of a grasp on this problem who has not read and reread the literature of two or more generations ago.

The infection gains entrance to the genital tract by way of the uterus or lacerations of the vagina or perineum, and may reach the peritoneum by contiguity without previous focal involvement of structures, but usually arises secondarily to some lesion where the invading organisms were momentarily halted.

How infections reach the peritoneal cavity is still not a matter of certainty. Usually the endometrium is the first structure invaded, but the infection rapidly spreads along the veins and lymphatics. My own limited observation would lead me to believe that the spreading infection along the lymph channels ultimately reaches the peritoneum. The more frequent venous thrombosis happily tends to remain localized. Even with all data at hand it is difficult to reach a conclusion in a concrete case because of the complexity of the picture. As an abstract problem one can scarcely do better than hide his ignorance behind a mass of statistics, being secure in the fact that the reader will see no clearer light than he sees himself.

I believe that there is something in the strain of bacteria carried from woman to woman in the old-time epidemics that made them so frightful. Their pathogenicity must have exceeded the ordinary one of virulent bacteria.

Diffusion through the lymphatics or escape through the tubes are probably the common sources as they are the most obvious

possibilities. Reaching the surface of the peritoneum the inflammation is rapidly diffused over the whole surface of it. That the dissemination of infection is rapid and diffuse is evident by the overwhelming character of the invasion, an intensity rivaled only by perforations of the duodenum.

Secondary invasion of the peritoneum is possible after the disease has become localized. Such secondary invasion occurs usually from the tube or a walled-off pelvic abscess, abscesses in the wall of the uterus being apparently less often the cause of such secondary infection. In some of the recorded cases an intramural abscess discharged through the umbilicus. It is conceivable that more may have failed to reach such a distant part and have given rise to a generalized peritonitis, but the literature is strangely silent of record of such a catastrophe.

Septic thromboses may involve the peritoneum by extension through the vessel wall and the overlying peritoneum. In several cases which I have observed at autopsy a generalized gangrene of the uterus and adnexa apparently preceded the generalized infection.

Dr. J. N. Jackson once presented me with a uterus, broad ligaments and ovaries which he had removed from a puerperal woman that were blue-black throughout. The patient recovered. A generalized peritoneal infection certainly would have resulted in a short time had the necrosed organs not been removed.

**Pathology.**—The earliest recorded observations on the pathology of peritonitis were made on the abdomens of women dead of puerperal peritonitis and the accounts of the old writers may be transposed to modern literature without loss of meaning. The thin flocculent exudate covering a reddened peritoneal surface, or even in some instances a dry shineless surface without exudate, has been described. In other cases the exudate is more purulent, supplying a more legitimate excuse for a comparison with lacteal secretions. The angles between the guts are often occupied by fibrinous flocculi which hold adjacent folds of gut in loose contact. Such is about all that is seen in the rapidly fatal cases. In the less acute, walling off may occur producing a localized abscess which is amenable to treatment or may become the source of subsequent mischief.

The older writings abound in anatomic descriptions that make the above description seem dull. The following from Hulme may be quoted as an example: "The belly was greatly swelled. The skin of the whole body was of a tawny or yellowish hue. Upon viewing the abdominal contents, the omentum was found greatly mortified. A yellow fetid liquor, with a mixture of pus, filled the pelvis, and floated among the intestines. The whole intestinal canal was distended with fetid air, but particularly the great flexure of the colon. A general inflammation appeared, scattered in various parts, over all the intestines. The stomach was not distended with flatus but lay concealed under the liver, which was of an extraordinary magnitude. It had pushed itself, as it were, high up into the cavity of the thorax and carried the diaphragm along with it, to which it adhered so firmly, in its whole convex surface, as not to be separated. In the right lobe was found a very extensive abscess. The gall-bladder was pretty large, and full of bile. The lungs were of remarkably small size, dense and livid; they did not adhere to the pleura." This description can easily be duplicated hundreds of times in the older literature.

The pleural cavities are frequently infected in puerperal peritonitis, and septic pneumonia or abscess often follows. Parenchymatous degeneration of the organs was very common. The "black vomit" so often mentioned likely meant hemorrhage into the stomach, the result of an infected gastric mucosa. Cerebral edema and septic meningitis receive frequent mention. Metastasis in the joints, particularly in the knees, I myself have observed in several instances and many are recorded in the literature.

**Clinical Signs.**—The general symptoms of puerperal peritonitis parallel those already discussed under the general head of symptomatology—all magnified to the *n*th power. It remains here only to discuss the variations and peculiarities.

**Chill.**—An initial chill is more commonly observed than in other forms of general peritonitis, obviously because the blood stream is so often invaded. Usually within one to three days after labor the patient is seized by a chill lasting usually from thirty minutes to three hours. The chills are usually decisive, even violent. There may be but one initial chill or they may be repeated a number of times during the course of the disease, particularly when pyemic

processes make up a large part of the picture. When chills are recurrent with regularity, constant search must be made for complicating foci of infection.

*Temperature.*—Preceding this chill or following it there is a rise of temperature. The temperature usually ranges high throughout the disease, but not infrequently is observed to fall at one or two periods of the day. If there are distinct remissions with or without chill localized foci should be anticipated.

*Pain.*—Early in the attack there is intense pain in the abdomen rivaled only by that observed in perforating ulcer. It usually begins in the pelvis but rapidly spreads over the whole abdomen. This pain is continuous but is increased by movement and by external pressure, not alone by the palpating hands of the observer but even by the weight of the bed clothes. The excessive sensitiveness represented in this type is not equaled by that due to any other cause. Sometimes there is but slight pain or an entire absence of it. This is apt to occur when there is preceding gangrene of the pelvic structures. Sometimes the initial intoxication is so overwhelming that there are no spontaneous complaints of pain, but when the abdomen is palpated reflex manifestations of pain are easily elicited.

*Tympany.*—If one has never seen a case of tympany in a puerperal woman he has no real conception as to how much the word may mean. Because of the laxness of the abdominal walls due to the pregnancy the distention is astonishingly extreme, exceeding much that of the pregnant abdomen. It is most marked when there is rapid diffusion of the infection. When there is deep involvement of the gut wall, particularly when the peritoneum is involved secondary to gangrene of some of the pelvic organs, there may be but slight if any tympany. I have never seen a scaphoid abdomen as one sometimes sees after appendicitis.

*Diagnosis.*—In a classical case the diagnosis is easy. The signs already enumerated can leave no doubt in the mind of the observer. It is only the milder, more slowly developing forms that can be compared with other postpuerperal infections. However, venous thrombosis may engender chill, rise of temperature and abdominal distention. Muscle rigidity is not the aid that it is in other forms of peritoneal infection. Because of the laxity of the muscles they



seem unable to lend the protective aid they do in other types of peritonitis. Here tenderness is the sign on which greatest reliance may be placed. When the initial signs above noted are associated with upper abdominal tenderness a generalizing peritonitis may be assumed. Tenderness over the pubic region is present in tubal and broad ligament infections. Indurated masses within the pelvis may be sometimes demonstrated and give the hope that the process may be localized. In lieu of any localized manifestations the breasts should be examined. The assumption on the part of the practitioner that delayed bowel movements may be the cause of chill presages an unstable state of his own system when the real truth dawns upon him. I know of what I speak.

**Prognosis.**—The final outcome of puerperal peritonitis is much more difficult to foretell than in other types of peritonitis because the diagnosis is never so clear cut. Even if one observes at operation what degree of involvement exists he can not estimate the influence of the possible complications which may exist or may subsequently develop.

That really diffuse cases ever recover surpasses belief. What degree of extension is compatible with recovery is impossible to say. Involvement of the pelvic peritoneum only, no doubt is often followed by recovery. The difficulty in forming an idea of the outcome from recorded cases lies in the fact that operators differ so widely in their interpretations. That a really diffuse case ever recovers I do not believe, no matter what the treatment. The proportion of cases of recovery following treatment is difficult to determine because the thromboses and extraperitoneal inflammations are not sharply separated from diffuse peritonitis. This is indicated by the writings of such masters as Leopold and Bumm. The former had 13 recoveries out of 18 cases treated. With the latter in a collected series of 177 cases 60 per cent recovered while in distinctly peritonitic cases 52 per cent recovered.

When complications exist they may cause the death of the patient after the peritonitis has subsided. Joint suppurations are particularly liable to exhaust the patient. Degeneration of the heart and parenchymatous organs also must be taken into consideration in estimating the outcome.

**Treatment.**—The results seem to be best if simple drainage with

large tubes in one or more regions is instituted early. Attempts to remove foci of infection seem unwise, though some good results have come from hysterectomy performed early in the disease. If localized abscesses occur they must be drained extraperitoneally. This is important because a postpuerperal abscess may remain infective many months, even years. I once lost a patient by trying to remove a postpuerperal streptococcic pyosalpinx three years after the infection. When the hyperacute type exists the human race will best be served if the attendant will go away and wrap his head in sackcloth and ashes.

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## CHAPTER XXIV

### TRAUMATIC PERITONITIS WITHOUT RUPTURE

External violence may so far injure an intestine that, while it is not immediately pervious to bacteria, it may become so after an interval. The passage of bacteria may take place through a microscopic opening, or they may pass through the injured wall without a demonstrable opening. In the interval between the receipt of the trauma and the escape of bacteria reactive changes may occur in the environs of the injured area, resulting in the formation of protective adhesions. If changes do not occur a diffuse peritonitis results. The perforation in such cases resembles the punched out ulcers of the duodenum.

**Pseudoperitonitis.**—In the literature one finds a number of cases reported in which there were acute abdominal symptoms but no real peritonitis. Possibly a retroperitoneal mesenteric hemorrhage or thrombosis of one or more vessels may account for such symptoms. Some of these cases present many of the symptoms of peritonitis, notably pain, distention, and vomiting. In some of these cases no pathologic lesion whatever can be found. I have observed two such cases. One, a carpenter, was struck in the pit of the stomach. He fell at once in a faint but soon recovered and complained of pain and vomited several times. Within two hours an astonishing degree of distention took place. The temperature did not change and the pulse, accelerated immediately after the accident, gradually subsided. Operation disclosed nothing. I always felt somewhat chagrined at having operated on this patient. Years later Fontoynt reported a case very similar to mine in which he searched very carefully and found nothing. I appreciate the feelings of the author just quoted when he states that as he searched he found always “vein, toujours vein.” The only thing I found of note was a very wide distention of all the veins of the abdominal organs. My second case, resulting from the kick of a horse, was very similar. I decided to sit tight until some signs of

reaction appeared—increased temperature or pulse. None appeared. Similar conditions are sometimes noted after injuries to the back. The condition probably results from some sort of an injury of the sympathetic system. At any rate it does not clarify the problem any to force them into the class of inflammatory lesions.

The number of posttraumatic peritonitides without rupture is not large but a number of cases have been reported. Guibal gives a collective review. In 26 cases, he notes 16 diffuse and 10 localized. Among these are a number, however, in which no inflammation was found at operation.

**Localized Peritonitis.**—After a trauma the contused gut becomes adherent to its environment. Increasing degeneration of the center of the lesion permits bacteria to escape into the area already walled off. An abscess results. The extent of this abscess varies greatly. It may rupture into the peritoneal cavity producing a diffuse peritonitis. It may remain localized and permit drainage or even drain spontaneously. I had such a case. A man was kicked in the lower abdomen by a horse. He had pain at once, followed by fever and distended abdomen. I saw him in 3 weeks and opened into a tumefied mass and found much gas and colon bacilli. A fecal fistula persisted for six months.

**Diffuse Peritonitis.**—In cases in which there is no reaction following the injury when the injured area becomes separated from the surrounding tissue the intestinal material escapes and a diffuse peritonitis results. This event is most apt to occur in cases in which the violence of the injury was not extreme. The injured area becomes necrotic, the result of thromboses, and drops out permitting the contents of the gut to escape with the usual results. These are very treacherous cases. I once saw a man who had suffered a moderate blow in the epigastrium. He had pain which soon subsided. He was attacked with acute abdominal pain a week later. An area the size of a dime had fallen out of the wall of the colon. The affected area while undergoing necrosis may give no symptoms before the lesion suddenly gives way. History of trauma then is the only fact that may aid us in distinguishing such cases from a peptic or other ulcer. When the escape of infection takes place gradually through an opening partly walled off, the onset

is less acute. The course of the disease is then similar to a non-perforating appendicitis.

**Diagnosis.**—When confronted by a questionable abdominal injury the patient should be placed in surroundings where immediate operation may be done should events demand it. The kind and degree of violence executed may give some clue as to what complication may be expected. The beginning symptoms may be those of distention with little spontaneous pain or pain on pressure. Superficial tenderness may be due to contusion of the abdominal wall. This should be at its height in 36 hours. Should it increase after this time a graver lesion must be expected. Should muscular rigidity increase an impending infection is probable. In such cases the pulse and temperature usually keep pace with the increase in the local reaction. If the progress is slow, one may be able to appreciate a gradually walling-off process.

The perforating type in which there is no evidence until the necrotic area separates presents no evidence which enables one to make a diagnosis until the perforation occurs. The occurrence of symptoms of acute perforation a week or so after an abdominal trauma should be the signal for action.

**Treatment.**—When there is evidence of internal injury of the character above described an exploratory laparotomy may be the safest course. One can not place his indications before the patient too strongly, for he may refuse operation and go about his business undisturbed. It is only the evidence of a progressive reactive process that warrants unequivocal advice of operation. With a slowly ascending inflammation with evidence of localization safety may lie in procrastination until the process becomes walled off. The procedure then resolves itself into the simple opening into a walled-off abscess, with the likelihood of producing a temporary fecal fistula. These usually heal spontaneously if they are located in the terminal ileum. Even if the fistula closes, the loop of gut likely will remain attached to the abdominal wall and these adhesions remain as possible sources of further mischief.

When there is a perforation through the gut wall as a secondary necrotic process the treatment usually accorded a perforating duodenal ulcer is in order. Usually the perforation will be be-

neath the great omentum and the drainage must be placed accordingly.

**Peritonitis from Traumatic Rupture of the Gut.**—Direct violence may sever a loop of gut resulting in the immediate escape of fecal contents with subsequent acute diffuse peritonitis. The nature of the injury, such as being run over by a vehicle, or by a blunt object as a wagon tongue, should excite the apprehension of the surgeon and if in a few hours the pulse should mount, exploration is demanded. The disease is an uncommon one and is usually overlooked until active inflammation has become advanced.

Laceration of solid parenchymatous organs with hemorrhage may irritate the peritoneum and give the symptoms of a generalized peritonitis. If the hemorrhage is extensive, early symptoms of anemia may give a clue to the correct diagnosis, but a small hemorrhage may cause a moderate rise of temperature, pain and general tympany.

**Rupture of the Mesentery.**—A solution of continuity of the mesentery as an isolated lesion is not common. Neumann reports a case. Kudlek reports two cases. Autenrieth reports one case and Erdman reports three cases. Hume reports one case as does Ogden.

The significant event in all these cases is hemorrhage. Pain and collapse in proportion to the extent of the leak follows. When there is traumatic rupture of the gut the mesentery is likely to be involved to a greater or less extent. Bruising of the mesentery may cause clotting within the vessels with subsequent gangrene of the gut. This is likely the etiology in many cases of late perforation of the gut already discussed.

## POSTOPERATIVE PERITONITIS

General peritonitis following a "clean" abdominal operation is to the surgeon what puerperal fever is to the obstetrician—a catastrophe. They rarely occur in the hands of skilled surgeons, but they do occur now and then even under the most favorable surroundings. When such tragedies do occur usually either the operator or some one of his assistants has come in contact with some virulent infective material. During the time I was an innocent laboratory worker I noted that when surgeons worked with

erysipelatous patients and like infections there was likely to be a call to do an autopsy on a patient dead of "paralytic ileus," whatever may be meant by that term.

**Prophylaxis.**—Two general plans have been followed: the systemic stimulation of the system to leucocyte formation, the leucophytaxia of Jousse, and a local increase of leucocytes confined to the peritoneum itself.

A number of substances have been employed to produce these results. The nucleinic acid and nucleinate of soda have been most studied. Von Mikulicz employed the former substance in normal salt solution by injecting it directly into the peritoneal cavity. He found that animals so treated resisted five times the lethal dose of colon bacilli. These animals also resisted the introduction into the peritoneal cavity of gastric or intestinal contents. Faucon in repeating these experiments found that the animals were not protected when virulent bacteria were added or the intestinal contents which were allowed to escape. In employing this substance clinically Mikulicz injected 50 c.c. of a 2 per cent solution.

Aschner and v. Graff employed the solution subcutaneously. They found that severe pain, requiring morphine, was caused as well as fever and general depression. All these phenomena were more intense in young subjects.

The clinical results of this practice are inconclusive. Jousse collected 1047 cases in which it was used. The operative mortality is alleged to have been lowered from 9.6 per cent to 4.6 per cent. As one reads these case histories it is difficult to suppress the feeling that the reputed inaccuracy of statistics here reaches the superlative degree.

To Glimm belongs the doubtful credit of first employing olive oil as a protective against postoperative peritonitis, though Brennen seems to have suggested that it may prevent the development of bacteria, this being his explanation of its alleged prevention of adhesions. Assuming that the oil, as well as bacteria and their toxins, are absorbed by way of the lymphatics, he proposed to lessen the deleterious effects of peritonitis by plugging up the lymph channels with oil globules. He found that rabbits in which 8 to 10 c.c. were injected into the peritoneal cavity were protected from fatal doses of colon bacilli. Hoehne in repeating these ex-

periments found that the animals were not so protected. He found that the oil is absorbed because he observed pulmonary embolism. Hoehne concluded that absorption of microbes was diminished if the oil is injected 12 to 24 hours to 4 days before the injection of the microbes. This slowing is due, he concludes, to reactive inflammation. He employed it in 42 patients with satisfactory results. Hirschel employed it in 3 cases of diffuse peritonitis and credits this treatment with having saved one of them. He later reports 9 more cases with 4 recoveries. In these he used from 100 to 300 c.c. of 1 per cent camphorated oil.

Petit used horse serum for the prevention of peritonitis. He found that this substance made rabbits resistant to 5 times the lethal dose of typhoid and colon bacilli. He reports good results in existent peritonitis. Schmidt found that the use of the serum produced a leucocytosis. Federmann employed it in 11 cases of general peritonitis with 3 recoveries.

Numerous other substances have been used. Lardennois used 20 c.c. hydrocele fluid intraperitoneally. Doyen employed fibrolysin. Weiss and Sencert employed oxygen. Finally Miramond de Laroquette employed ozone by means of the x-ray.

So far as concerns the value of the means above enumerated as a postoperative prophylactic, the perfection of modern technic makes them entirely superfluous. As therapeutic measures they illustrate the fact so often noted in the discussions of treatment that an author makes enthusiastic reports of the value of certain measures then promptly ceases to employ them in his own practice. Though each of the substances discussed had its enthusiastic supporters, none are used at the present time.

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## CHAPTER XXV

### FETAL PERITONITIS

Under this heading a great variety of intraabdominal conditions as observed in the fetus have been described. A few cases of real peritonitis have been recorded, but most of the literature relates to unusual developmental conditions either pertaining to unusual union of peritoneal surfaces or to so-called adhesion formations.

Orthmann discusses particularly the relation of fetal peritonitis to double uterus and vagina duplex. Von Winkel also ascribes many developmental anomalies to fetal peritonitis. Both these authors discuss the literature.

Simpson through Bednar was able to report on a collection of 186 cases. Therein brings the records to the date of his paper and muddies the waters by declaring that all gut occlusions of unknown origin must be attributed to peritonitis, whether or not there is evidence of the former existence of such a process. Ahlfeld attempts to get away from this broad assumption by accusing the prolonged persistence of the omphalomesenteric duct. Silberman brings the study to the date of his paper, reviewing fifty-seven cases from the clinical viewpoint. He notes that twenty-four of these occurred in the duodenum, permitting us, in the light of recent study, to bring the problem rather automatically to date and to rid it of the confusing designation "inflammation" and removing it from the scope of this monograph.

The reason "peritonitis" is invoked as a cause of anomalies is that adhesions, ascribed to previous inflammation, are supposed to interpose between the developing organs. The fundamental error in this assumption is that the term inflammation is assumed to be coextensive with pathologic process. What the cause of these unusual conditions may be, we can not speak of with certainty, but no one has ever demonstrated an inflammatory state in any of the specimens recorded.

The conditions described as being due to adhesions are analo-

gous, most likely identical, with normal adhesions, that is to say the normal obliteration of surfaces which takes place when a surface once intraperitoneal becomes extraperitoneal. Why normal adhesions take place can not of course be definitely stated, but that the abnormal is but an extension of the usual can not well be doubted, because of their like histologic appearance and the fact that every gradation from the normal can be observed.

Dalla Rose questioned the justification of ascribing these adhesions to postinflammatory processes, preferring to refer to them as developmental anomalies. As already noted none of the phenomena of inflammation precedes their formation and the use of this term merely confesses the paucity of our nomenclature. These changes were noted as early as the fourth month in two cases and between the sixth and seventh month in two other cases by Simpson. Orthmann places them at the sixth to the eighth week, Gessner at the third month, and Mackenrodt at from the third to the sixth month. It seems worthy of note that in these young fetuses adventitious bands are vastly more common than at term. Conclusions based on such observations are apt to be erroneous for in the further development these "bands" must be used up by the expanding organs. Many of the "bands" in these young fetuses when sectioned fail to show an organic structure and they may wholly disappear, judged by the standards we may say that they would disappear.

In view of the available facts it seems impossible to define a limit between unusual development of normal processes and the pathologic. It seems safer in case of doubt to ascribe such occurrences as an excess of the normal, being pathologic only when there is an interference with the proper performance of the function of the organs of the abdomen. The term "peritonitis" had best be reserved for those few instances in which there is evidence of a reactive process. At the same time it must not be lost sight of that membraniform agglutination may occur in the adult as a result of irritation processes which do not lead to suppuration. Some of these we may ascribe to tuberculosis. Possibly this organism may be active in the pelvis. These processes are in need of renewed study.

It may be noted that the gut tract being free from bacteria, any disturbance in the circulation capable of producing an exudate

might be followed by organization of the exudate. It still remains for someone to advance the theory that the attachment of the colon is due to circulatory disturbance due to the rotation of the gut.

The source of origin of the infection in these alleged peritonitides has been hypothesized as being derived through the maternal circulation as Peiser has pointed out. That such a transmission is possible has been shown by Blumenthal and Hanne for the colon bacillus and by Neuwerck and Flinzer for the paratyphoid bacillus. Still it is a far cry from the finding of bacilli in the fetal blood and to the cause of adhesion in the peritoneal cavity.

The anatomic findings usually described in cases of fetal peritonitis are described as exudates in which fibrin flocculi are suspended, or sometimes fibrinoid precipitates more or less intimately attached to the peritoneal surface which may agglutinate peritoneal surfaces together as in a case recorded by Baumgarten.

It is not uncommon to note a coagulable exudate in the bellies of fetuses. The agglutination is purely due to coagulable material and there is no attempt at organization, neither is there any reaction on the part of the peritoneum. Why this exudate forms is not known. Whether it bears any relation to the equally mysterious hydrocephalus or hydramnios is a matter of great concern. These exudates are sterile and occur independent of any infective process of the mother. Possibly impending abortion interferes with the placental circulation and the peritoneal exudate is but a dropsy due to passive hyperemia, analogous to dropsy in liver cirrhosis. The absence of a cellular content in both would make such an explanation as logical as others that have been advanced.

Contrasted with the intangible conditions above noted are those arising secondary to other conditions. Peiser has divided these into those arising from congenital atresias and stenoses, torsion of the gut, perforation of the gut and maldevelopment of the urogenital apparatus.

There is a group of cases in which there is evidence of past inflammation. In most of these a cause for the inflammation has been demonstrated. The first group of causes is congenital anomalies, chiefly atresias, inflammation being in these cases the result, not the cause of fetal peritonitis. The other most frequent cause of fetal peritonitis is necrosis from inspissated gut contents.

In most of the bona fide cases in which peritonitis was associated with atresia there was evidence that stasis occurred behind the constriction of the gut. Perforations may take place in the absence of atresia, however. Why these perforations take place is not known. Paultauf believed they were due to stercoral necrosis. Schlengel records a pea-sized perforation near the ileocecal valve. Generisch reports a meconium abscess. Zillner believes rupture of the gut may take place during birth. Thus Brandfoot records a case in which there were organized adhesions in the right iliac fossa of the small and large intestine. Ballentyne's case was a child which died 32 hours after birth which showed extensive dry adhesions. The oldest process seemed to be in the pelvis. Generisch's case died 45 hours after birth. The abdominal wall was thickened and was infiltrated with a thin cloudy greenish serum. The intestines were agglutinated with each other and with the abdominal wall. There was an opening in the ileum 10 cm. from the cecum which communicated with a cavity formed by adherent gut and abdominal wall. Falkenheim and Askanazy reported a case in which there was a perforation of a gut by calcified meconium.

Most of the cases reported, judging from the state of development of the gut, seem to have arisen at the end of pregnancy. Gessner believes his case originated in a peritonitis because the intestines were enveloped by a thin membranous adhesion which extended over the liver.

I once studied a stillborn child which had the intestines matted together by a coagulated gelatiform mass. Near the sigmoid colon the small intestines were matted together by membranous adhesions. Near the pelvic brim the sigmoid made a high loop in which was a mass of hard meconium which had much thinned the gut wall. About this point the small intestines were adherent to the sigmoid.

Owing to the sterile character of the intestinal contents its escape into the peritoneal cavity produces only a chemical peritonitis. As observed they are mostly end-products. One case of suppurative peritonitis was reported by Hunt. This patient had lived but an hour after birth. There were old adhesions and the omentum and intestinal serosa were hyperemic and were covered

with pus. This seems to be the only case recorded in which "pus" was noted.

As examples of the circumscribed type which has run its course may be mentioned the following by Theremin. In this case all that remained at autopsy was a contracted loop of ileum the mesenteric borders of which were firmly adherent. Similarly Dorn reports a case in which the duodenum was much contracted and there were adhesions between the duodenal wall, colon, and neck of the gall bladder to the right kidney. Peiser reports a case in which there were diffuse adhesions in a child six months old. There were membrane-like adhesions over all the viscera and the pelvis was wholly inaccessible because of them.

Despite the indefinite state of our anatomic knowledge Noetterbrock discusses the possibility of a clinical diagnosis. He thinks that there is a possibility of feeling the distended abdomen of the fetus in cases when the liquor amnii is small in amount, also in breech presentation the absence of expulsion of meconium might indicate a stenosis. It is worth noting that the proposal of making a clinical diagnosis was made in a student's thesis.

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## CHAPTER XXVI

### TUBERCULOSIS OF THE PERITONEUM

**Historical.**—The foundation upon which our conception of tuberculous peritonitis is builded was laid by Bichat, who separated the inflammations of the peritoneum from the diseases of the stomach and intestines, and divided them into acute and chronic forms. The chronic form, which concerns us here, was not separated from other chronic diseases, notably carcinosis. It is true, Morton, Bonet, and Morgagni had previously written more or less accurate descriptions of the disease, but Baille had not yet clearly defined the tubercle as the specific lesion of tuberculosis. The recognition of a distinct type for tuberculosis remained for Baron and, following him, Louis. The latter noted the identity of the lesions existing in the peritoneum with those in the pleura in pulmonary phthisis. Broussais predicted its general recognition. It is interesting to note that Hodgkin opposed this view.

Louis believed that peritonitis was always secondary to pleurisy. Godart opposed this view, for he observed a case in which the lungs were unaffected. Bright, Trousseau, and others noted similar cases. Louis's observations were further limited by Cruveilhier, who noted cases of chronic peritonitis in which there were no tubercles. Since this occurred mostly in young women, he called this type "*Ascite des jeunes filles*." This conception was further developed by Aran, and this form was subsequently recognized as idiopathic chronic peritonitis. For a time this idiopathic type prevailed as the more common one. The relation of this type to the tuberculous form is perhaps today not fully determined. Aran observed that chronic peritonitis was noted chiefly in tuberculous subjects and that in such cases tubercles were usually found, but he observed cases in which chronic peritonitis occurred in those not affected by pulmonary tuberculosis.

A new era in the history of tuberculous peritonitis began in 1884 when König recounted case histories in which the patients



recovered. With this announcement, the modern history of tuberculous peritonitis may be said to begin. The importance of this announcement was not simply that a therapeutic measure of importance was introduced, but that it opened up a vast field for the observation of the disease in its early stages and for the clearing up of the diagnosis in many cases. From this time on the history merges into the account of its pathologic anatomy.

**Etiology.**—The cause of tuberculous peritonitis is, of course, the tubercle bacillus.

The determination of the specificity of tuberculosis is a matter of interest. While Louis recognized it as a local manifestation of a general disease, it was Villeman who first announced the theory of the infectious character of pulmonary tuberculosis. Before the discovery of the tubercle bacillus by Koch, in 1882, many writers had surmised the tuberculous nature of certain lesions of the peritoneum. The conclusions were based, of course, on the recognition of the unit lesion, the tubercle, reasoning by analogy from similar lesions in the lung.

It is important to note that in the determination of clinical problems we still employ the results of the observations made in the period before the discovery of the bacillus. These conclusions were based on the presence of tubercles, with infiltration and caseation. Even with the aid of modern technic the demonstration of the bacillus in a given case of the disease is often difficult, even impossible, either in the exudate or in the tissues, in cases in which the gross appearance of the lesion and the clinical course leave no doubt as to the nature of the disease. There are cases of chronic exudative peritonitis which lack the typical anatomic lesion of tuberculosis, but which, because of their chronicity, resemble tuberculosis. Prior to the discovery of the tubercle bacillus, these cases were classified as idiopathic. Since the discovery of the bacillus this group has been markedly reduced. Many observers even deny the occurrence of idiopathic cases. Be this as it may, chronic peritoneal inflammations in which it is impossible to demonstrate the bacillus, do occur. It would, perhaps, be better to classify such cases as idiopathic, and to be more rigid in our demands for scientific proof of their tuberculous nature, than to categorically force them into the specific group. The question is of

sufficient interest to warrant a separate discussion of idiopathic peritonitis.

When the specific cause of a disease is known, the etiologic factors available for discussion are confined to a recounting of facts pertaining to the conditions and circumstances under which the disease develops.

**Age.**—No age is exempt. Monclair and Alglare have reported a case of a baby which died on the sixth day after birth. The autopsy showed an extensive tuberculosis of the peritoneum. The mesenteric glands showed giant cells and tubercle bacilli. Bouardel reported a case in an infant of ten weeks of age. Fletcher records eight cases under one year of age. Cummins' statistics show a variation in age from sixteen months to seventy-three years. In König's series 30 per cent were below twenty years. Thoenes found two-thirds of the cases in young persons. Osler, however, states that the disease occurs most frequently between the twentieth and the forty-ninth years, and this corresponds to the statistics of most authors. Thus Nothnagel had 83 out of 164 cases between the twentieth and fortieth years, and Shattuck had 52 out of 98 cases during the same period. Schmalmack found the ages most predisposed to be between one to ten and twenty-one to fifty years.

**Sex.**—The sex incidence varies according to whether postmortem or operative statistics are considered. In the former the male, in the latter the female predominates. Thus König in 131 cases operated on found only 11 in men, while Rosthorn in 153 autopsies found 122 in the male and 31 in the female. Fenwick in 46 cases had 30 males and 16 females. Philipps in 107 postmortem cases found the male affected 89 times, and the female 18 times; in operative cases, he found 120 females to 14 males. Münstermann in 46 operative cases had 33 females and 13 males; Cummins had in 92 autopsies 31 females and 61 males. Voigt had 29 females and 27 males in 56 cases. Taking this large series, we find that about three times as many males are affected as females. Schmalmack suggests that the reason for this discrepancy is due to the fact that more males than females come to autopsy, and more females than males to operation. This suggestion is a good one. Heintze suggests, as the cause of greater frequency in males, that alcoholism

predisposes to tuberculous peritonitis. Alcoholism at least predisposes to death in a public hospital, with a subsequent autopsy and a correct diagnosis. In either event the statistics are swelled in favor of the male. Numerous writers, for instance, Rokitansky, Förster, Weigert, and Grawitz, however, suggest that hepatic cirrhosis predisposes to peritoneal tuberculosis, and in this way alcohol might play an indirect part. On the other hand, it is pointed out (Heintze) that the pelvic congestion of puberty, hematocele, and pelvic peritonitis, predisposes the female to this disease. Allport believes that the percentage of sex incidence would be equalized "were we to add to the percentage of male operated patients those who suffer from tuberculosis of those organs whose female homologues are intraabdominal; or were we to deduct from the percentage of laparotomized women those who suffer from secondary or ascending tuberculosis of the intraabdominal genitals and tuberculous peritonitis incident thereto." It is a general agreement that the genital organs are the point of beginning in the majority of cases in the female; but whether this is true or not is a question. That so few females, relatively, come to autopsy may be explained on the ground that many recover spontaneously, inasmuch as the discrepancy of sex at autopsy was noted before the pelvic organs of the female were so often attacked by the surgeon. This fact should be remembered in considering treatment.

**Heredity.**—A family history of tuberculosis is obtained in but a minority of the cases. Heintze obtained such a history in four out of twenty-five cases. Häne found a positive history in 35 per cent of cases. Delpuech states that a hereditary history is exceptional.

**General Physical State.**—The general conditions under which tuberculous peritonitis develops are parallel with those underlying the disease in other organs. This is obviously true since in the vast majority of cases, the disease first finds a nidus in some remote organ, notably the lungs.

Maldevelopment, bad housing conditions, and insufficient nutrition because of poverty and chronic disease of the digestive organs are said to play a part. Maurange regards an alcoholic or syphilitic heredity as an important predisposing factor. Arullani considered drinking a great factor in the production of all forms of tuberculosis. Vallin noted this disease most frequently among sol-

diers. My experience is in accord with those observers who see peritoneal tuberculosis attack those who have no tuberculous history and who were previously healthy.

Whether some preexisting affection of the peritoneum or its immediate environs presages the advent of peritoneal tuberculosis is still a matter of speculation. Thus Boulland believes that a scar resulting from typhoid or other ulcerations may have an etiologic importance. He also regards the congestion of menstruation and the hypernutrition of pregnancy as factors which favor the development of the disease in these organs. Steinbrücke accuses chlorosis of having a deleterious influence. Delpuech sees in irritation and preexisting inflammation an important predisposing cause. That irritation does play some part, would seem to be indicated by the repeated localization in hernial sacs, as already noted by Cruveilhier.

**Trauma.**—In a number of instances a trauma has preceded the development of the disease. Broussais regarded trauma as an important factor. O'Callaghan reports a case which developed in a boy after an injury received during a football game. Plummer reports a case following a kick by a horse. I had a case in a male, aged forty-six, in whom a cystic mass developed in the umbilical region, following a violent injury. There was rapid emaciation, and pancreatic cyst was diagnosed. Operation disclosed an encysted tuberculosis. A number of like cases are reported in the French literature.

Kelly believes there is a definite relation between pregnancy and tuberculous peritonitis. In 28 per cent of his cases the disease dated from childbirth, and Boulland believes the frequent congestion due to menstruation and pregnancy favor the development of tuberculosis.

**Pathogenesis.**—While in rare instances, probably, the peritoneum may be the site of the initial lesion, in the vast majority of cases this organ becomes infected from a primary focus located elsewhere in the body. Such a focus can often be located in the lungs, lymph glands, Fallopian tubes, etc. In other instances it can only be suspected from the previous history. Even when the primary focus is known, the avenue by which the tubercle bacilli reach the peritoneum can not be demonstrated in the vast majority of cases.

The results of autopsies are uncertain, because the lesion is then advanced, obscuring all traces of the initial process. The evidence obtained at laparotomy is often inconclusive, for investigation is necessarily limited. Even in cases in which conclusive evidence of the existence of a primary focus can not be obtained, thorough attempt to find such a focus should always be made. Although a primary focus is known to exist, the avenue by which the infection reached the peritoneum can not be demonstrated. There is a huge literature on the subject, but there is still a great lack of definite knowledge on this phase of the subject.

The investigator should always seek to answer the question whether the peritoneal infection is primary or is secondary to some other focus. If secondary, the route traveled to reach the peritoneum should be determined, if possible.

**Primary Form.**—The limitations in our ability to satisfactorily demonstrate the genesis of a tuberculous peritonitis are particularly impressive when an attempt is made to answer the question whether it is possible for tubercle bacilli to reach the peritoneum without having previously involved some other organ. Rokitsansky commented on the extreme rarity of such an occurrence, and most of the later writers have not committed themselves definitely. Allport is quite certain that there is no such thing as an absolutely primary tuberculosis of the peritoneum. He denies categorically the transmission of bacteria across the normal intestinal wall. With this opinion there can be no disagreement, but an associated enteritis might produce a sufficient lesion of the gut wall to permit the passage of bacilli, just as the ileum permits the passage of colon bacilli through the damaged intestinal wall in strangulated hernia. The possibility of transmission from a focal infection, such as the tonsils or lung, by way of the blood stream can not be denied. Primary peritoneal tuberculosis can be produced by the injection of tubercle bacilli into the mesenteric arteries of animals, and the possibility of the passage of bacilli direct to the peritoneum after absorption from the tonsil can not be denied, for Lexer proved this possibility in case of the pus organisms. However this may be, primary peritoneal tuberculosis has not yet been proved. Children affected with tuberculous lymph glands may develop tuberculous peritonitis spontaneously or after some other disease. I have twice

observed a rapid development of tuberculous peritonitis in such children who had recently suffered from measles. The diagnosis was established by biopsy. Both of these recovered, and no secondary focus was ever discovered. Each had a tuberculous history, however, and presented general evidence of a substandard physique. It is likely that the exanthematous affection lighted up a dormant focus in some distant organ. To explain the advent of the infection in the peritoneum, it is necessary to assume that it was transported by the blood stream. The relative immunity of the peritoneum to tuberculous infection, to which Weigert called attention, would leave unexplained the escape of vulnerable organs, if the bacilli traveled by way of the blood stream, unless we assume that in certain instances, as from previous disease or otherwise, the peritoneum in a given case was particularly susceptible. In this regard the peritoneum bears much the same relation to the tubercle bacillus as it does to the pneumococcus.

I have seen several cases of tuberculosis of the cecum in individuals otherwise clinically free. In these cases the subserosa seemed to be the area chiefly involved. I had one patient, a boy of fourteen, who became suddenly sick with a moderate attack of acute peritonitis. At operation a moderately inflamed cecum and appendix were found. There was a solitary lymph gland in the mesentery which was removed. On section it showed the typical lesion of tuberculosis. The appendix showed only a general infiltration with acute necrosis in some of the lymph follicles. Since the gland removed was the only one affected it seems probable that it must have received its infection from the gut. Possibly the cecum was the primary seat of disease, a condition the appendix did not show.

I have seen a circumscribed peritonitis follow a trauma in a vigorous man of forty. While there was no obvious lesion, it is fair to assume that there was one.

The cases that appear primary at the operating table are most likely intestinal in origin. It is generally recognized that the retroperitoneal lymph glands are frequently infected. These must receive their infection from the gut mucosa. It is quite reasonable to assume that a lesion here will sometimes approach the serous surface. In the cases I have seen in children most of them were

under par in the year preceding the development of their peritoneal affection. I believe if pot-bellied children were examined more carefully for free fluid in the peritoneal cavity one would be led to suspect peritoneal tuberculosis more often. Old adhesions and vascular changes observed in later life may find an explanation in such a past process. Herringham found lesions in 50 cases in which tuberculosis was suspected, but could not be proved. Many such mild lesions could easily escape recognition. When the lesion in the peritoneum is once established, it is difficult to say which is primary when the lymph glands are also involved. While the origin of the lymph gland tuberculosis from the intestinal mucosa is largely hypothetical, the absorption from the intestine by these glands seems most likely. On the other hand Borchgrevink believes that the lymph glands in tuberculous peritonitis are usually free, escaping infection apparently both before and after the peritoneal involvement. The literature on this point is unsatisfactory.

Buszard reports a case in a man, aged 40, in which the suprarenals were evidently the primary seat of the disease. At any rate, the symptoms of Addison's disease preceded those in the peritoneum. Tuberculosis of the adrenals is of course relatively common, but there seems to be little tendency to involve the peritoneum.

The Fallopian tubes are apparently the most frequent primary seat of tuberculosis, judging from operative results alone. Mayo had 26 cases. Osler estimates that 30 to 40 per cent are primary in the tube, and Konlich places it at 71 per cent. Hanot believes that the tubes are secondarily affected from abdominal tuberculosis. Pinner's experiments show that the open fimbria may admit infection from the general peritoneal cavity.

At any rate, primary peritoneal tuberculosis is at least relatively infrequent. Obviously a primary lesion could be proved only in the rarest instances. Only complete autopsy could demonstrate such a condition with any degree of certainty. Even then, the preexistence of a focus elsewhere could not be denied, since before such an extensive study would be possible the process within the peritoneum would likely have advanced to such a degree that an origin by direct extension could not be excluded. Only in instances in which the patient should die early in the disease from

some other affection while the peritoneal involvement was yet in its incipency, could such origin be demonstrated with satisfactory certainty. The failure to find a primary focus at operation is no evidence that none exists. McNutt, for instance, records four cases as primary because they recovered, and no other focus was discovered. Such a conclusion is obviously unwarranted; nevertheless, patients without previous ill health often develop a peritoneal tuberculosis. I have been particularly impressed with the generally accepted opinion as to the frequency with which tubercles are found in the Fallopian tubes of otherwise healthy women who remain healthy after the offending tubes have been removed. A closer study of such tubes has made me skeptical about their tuberculous character in many of the reported cases. Tuberculosis is a virtuous disease, and charity may cause us to form such a diagnosis in some instances. The possibility of the tubercles being due to foreign bodies, from hemorrhage, or infection of the tubes, must be seriously considered. Necrotic foci cause the formation of foreign body giant cells which sometimes resemble tubercles; small sub-peritoneal cysts not infrequently are pointed out in the operating room as evidence of tuberculosis. Unless the actual tuberculous nature of the lesion is proved, either by demonstration of the bacilli or by an animal culture, I should be extremely guarded in the diagnosis.

**Secondary Form (Extension from Other Organs).**—Because of the rarity of the primary form, it must be assumed that the existence of peritoneal tuberculosis presupposes, at least in nearly every case, the existence of a tuberculous lesion elsewhere in the body. The problem to be solved is to find the focus and to determine how the infection travels from this point to the peritoneum.

In order to approach the problem with intelligence, it is necessary to consider the relative frequency of peritoneal tuberculosis and the frequency of the disease in other organs. The most instructive statistics from the American point of view are given by Cummins. In 3,405 autopsies collected from the Pennsylvania, Philadelphia, and University Hospitals, he found some form of tuberculosis in 835, or 24.5 per cent of cases observed. In addition 76 showed healed foci. Tuberculous peritonitis was found in 92 autopsies, or 2.7 per cent of the total number, or 11 per cent of



the tuberculous cases. Borschke's statistics show a still greater percentage of involvement. In 4,250 autopsies, 1,390 showed tuberculosis, and of these, 226 showed peritoneal involvement. Steiner found the peritoneum involved 92 times in 800 autopsies. The highest of all are the statistics of Nothnagel, who noted peritoneal involvement in nearly a fourth of his autopsies. Sick, on the other hand, noted a lesser proportion, and in 2,500 autopsies found no case of isolated tuberculous peritonitis, and in but 46 cases was the peritoneum affected at all. Münstermann in 2,837 autopsies found that 903 showed tuberculous lesions, and of these 46 cases showed involvement of the peritoneum, two of them apparently primary.

It may be assumed, therefore, that the peritoneum is involved in approximately 10 per cent of cases when death is caused by tuberculosis of some other organ of the body.

The lungs, naturally, are the organ most frequently affected. König found them involved in 92 per cent, the gut in 74 per cent, the kidney and spleen in 35 per cent each, the suprarenals and liver in 5 per cent each, and a generalized involvement in 10 per cent of cases of tuberculous peritonitis. Sick found the lungs involved in 85 per cent, the gut in 65 per cent, the genitals and liver in 26 per cent each, the pleura in 25 per cent, the mesentery in 20 per cent, and the kidneys in 19 per cent. Cummins found the lungs involved in 84 per cent, the gut in 32 per cent, the tubes and adnexa in 40 per cent, and the urinogenital organs in 8 per cent. Borschke, as already noted, reporting on postmortems of 1,393 tuberculous patients, found the peritoneum involved in 226, or 16.2 per cent. This author makes a distinction between tuberculous peritonitis and tuberculosis of the peritoneum. In the latter class he puts cases in which there is no reactive inflammation. Of this group there were 16 cases. These were for the most part without exudate, and but few showed any adhesions. In eight of these the infection involved only limited areas of the peritoneum. In the remainder of his autopsies the peritoneum was thickened with fibrinous or hemorrhagic inflammation. Over the tubercles was more or less fibrin. The great omentum was more or less involved, being indurated or contracted and rolled up by the distended intestines. Two of his cases were idiopathic. In 200 cases, the primary

affection was in the lungs. The unsatisfactory state of the available data can be noted by contrasting the statistics of Borschke's with those of Friedländer, above quoted.

Weigert notes in his paper that the relation of the peritoneum to tuberculosis is the same as to any mycotic disease and to tumors.

Klebs believed that the intestinal tract in children is the common avenue of infection. The chief argument in favor of this view is the relative frequency of mesenteric glandular tuberculosis in children. He assumed that the bacilli could gain access to the lymph stream or blood stream, and then gain a foothold in any part of the body, escaping the lymph glands and involving the peritoneum primarily. This would explain, but not prove, the origin of an isolated peritoneal involvement. Some more recent writers look favorably upon this possibility. Among these may be mentioned Trabaud, Straus, and Gamaleia, and particularly Lévi-Sirgugue.

The frequency with which intestinal tuberculosis is associated with peritoneal tuberculosis is difficult to determine from available statistics. The coexistence is frequent enough, but in most of the instances there is likewise an advanced pulmonary lesion. Cruveilhier laid stress on the importance of gut ulcers, and more recently König noted the coexistence in 80 of 107 cases. Spillman noted this association in 70 of 100 cases examined by his student, Colman. Schmalzmack noted that in all of his 19 cases in the male there were ulcerations of the gut and advanced lesions in the lymph glands. Delpuech believes that the intestinal lesion is usually secondary, because the ulcers are often recent, while the peritoneal affection is in the process of healing.

There can be no doubt that there are variations in the relative frequency of peritoneal tuberculosis in different localities as compared to the general morbidity of the population. Thus Dörfler in collected statistics of 80,000 cases of tuberculosis treated, found an involvement of the peritoneum in only 1.07 per cent of the cases. Borschke, on the other hand, in statistics of 1,393 cases, found 16.5 per cent with peritoneal involvement among the tuberculous cases. In the combined statistics collected by Biroher in 19,184 autopsies the peritoneum was affected in 3.5 per cent of the cases.

I dare say that in the average American community there is no such frequency as noted in the above statistics. In the community

about Halstead, Kansas, where I have been in close touch with the great majority of the population for more than twenty years, but few cases of tuberculous peritonitis have been noted. Conservatively speaking, some 100,000 morbid conditions have been prescribed for by me or one of my assistants, and but four cases of this disease have been found. At the Halstead Hospital, in some 5,000 examinations, representing patients from central and western parts of Kansas, for the most part, but two additional cases have been discovered. Tuberculosis in any form is a rare disease in that community, however, representing not more than 1 or 2 per cent of the patients examined.

The statistics above quoted are sufficient to indicate in which organs the primary focus occurs. In some instances we can only surmise the avenue of transportation. It can be easily proved experimentally that surface diffusion takes place, that is to say, bacilli gaining the surface of the peritoneum are transported by the movement of the fluids, aided no doubt by peristalsis, so that simultaneously foci appear in the various parts of the abdominal cavity. The same is true in the pleural cavity.

When the primary focus is known, the advent in the peritoneum may be accounted for by one of the following paths; by the blood stream, by contiguity, or by continuity (Allport.)

**Hematogenous.**—In instances in which the peritoneum becomes involved as a part of a generalized miliary tuberculosis, as after operations on tuberculous joints or glands of the neck, hematogenous transportation can hardly be doubted, since the lesion begins simultaneously in all parts of the body. In instances in which the primary focus is located in some distant organ, as in the mediastinal or other lymph nodes, with no intervening foci, the evidence is nearly as conclusive; but in the majority of instances the problem is not so simple, since intervening foci exist. In primary tuberculous peritonitis, if there is such a thing, transportation by way of the blood stream must be assumed. This is the easier, because the tubercles are often arranged along the course of blood vessels.

**By Contiguity.**—How the infection travels by this method from the primary lesion to the peritoneum may be indicated by the approach of the infection from the lungs to the pleura. Here the extension may be traced along the lymph vessels from the lesion in

the lungs to the pleura. This extension in the lymphatics may take place by a direct extension from one lesion to another until the pleura is reached, propelled more or less by retrograde metastasis due to the central occlusion of the lymph vessels by the central tuberculous lesion, as suggested by Ponfick. Friedländer found in 88 autopsies a local lesion in the neighboring tissue, and there was no occasion in any of them to hypothecate a hematogenous origin. These lesions, according to him, are most apt to lie in the intestinal submucosa, the lymph glands, or lymphatics. As these nodules approach the peritoneum, induration, possibly the formation of adhesions, takes place. These observations show plainly the likelihood of a combination of extension by contiguity and continuity. Contiguous lesions may coalesce, and a continuous lesion result. König compares the spread of the tuberculous process from contiguous organs to the peritoneum with that of the extension from the epiphyses of joints to the synovial surfaces.

**By Continuity.**—By this method is understood the direct extension from the primary focus to the peritoneum by continuity of disease without the intervention of any uninfected tissue. The most frequent example is the involvement of the peritoneum by direct extension through the walls of the Fallopian tubes. Extension from intestinal ulceration occurs less frequently or at least is less various. Whatever may be the truth as regards frequency, there is no doubt that direct extension is extremely important in a practical sense, because of the possibility of surgical removal of the lesion.

In many instances, even when primary direct extension can not be demonstrated because of the advanced state of the process, the relative degree of localization makes origin from near that point probable. The relative age of lesions also sometimes gives an idea as to the source of the infection. This is true most frequently in the case of the Fallopian tubes. Here it is not unusual to find an old caseated mass in the tube, surrounded by adhesions, and beyond this possibly a crop of young tubercles. Here the point of origin is definite, but how the organism gained this point can not usually be determined. After bacilli have gained access to the peritoneal surface the dissemination takes place by diffusion and by gravity. The lesions are most abundant in the dependent por-

tions of the abdominal cavity where inanimate bodies, such as lamp-black, are wont to collect in experimental work on animals.

The extension from the pleura to the diaphragm may be by direct extension or by way of the lymphatics. Lévi-Sirugue found lines of caseation extending across the diaphragm at autopsies. Valemin states as Codelier's law, "When there is a tuberculosis of the peritoneum, there is always a like affection of the pleura." Maurange believes that the primary focus of both of these conditions is in the lymph nodes of the mediastinum, and that the dissemination is by way of the lymphatics.

The length of time required for a tuberculous peritonitis to develop is probably not very great. I have seen a diffuse involvement within six weeks after stirring up a primary focus in an epididymis. Holmes reports a case of operation for a distended gall bladder, during which it was not noted that the peritoneum was free. Autopsy five weeks later showed the peritoneum to be everywhere involved.

**Pathologic Anatomy.—General Considerations.**—The genesis of the unit lesion, the tubercle, does not differ when located in the peritoneum from that situated elsewhere. The pathologic anatomy of the tubercle has long been established and one must resort to the older literature in order to find descriptions of it in the most graphic types. More modern pathology has established the histologic picture. More recently Smyth has added another stage in a study of the early stages of the tuberculous processes by observing it in tissue cultures. His work materially supplements earlier observations. He found that when cultures were inoculated they were at once surrounded by lymphocytes. Endothelial cells later reached the scene, and by conglutination formed the giant cells. His studies confirm therefore the conclusions arrived at by histopathologic processes. Modern studies have added little to the older views, but they have done much to strengthen our faith in the correctness of the older opinions.

**The Tubercle.**—The unit lesion of tuberculosis, the tubercle, received its name before its cellular structure was understood. It received extensive study by the naked eye. The frequency with which experienced surgeons confuse disseminated carcinomata with diffuse miliary tuberculosis makes me feel that the recognition of

the tubercle by the unaided eye is an art lost that may be deplored. On the other hand, our conception of the disease must be based on the finer changes which are revealed only by microscopic and cultural methods. It behooves the surgeon, therefore, to study the tubercle from both these points of view.

*Macroscopic Appearance.*—The name signifies a nodule. Bailli seems to have been the first to apply this name to the unit lesion of tuberculosis. He describes its appearance in the following words: "They are small grayish granules, semitransparent, sometimes transparent and colorless, and of a consistency a little less than that of cartilage; their size varies from that of a millet seed to that of a hemp seed; in form they are oblong at first glance but are less regular when examined with a magnifying glass, when they sometimes appear to be angular; they are intimately attached to the underlying tissue and can not be separated from it without causing shreds of tissue to follow it."

It is desirable to remember that even these small lesions are not coextensive with the tubercle in the microscopic sense, but are made up of a number of these. Here is the clue to the irregularity Louis noted. When secondary nodules form they appear "angular." The cause of their oblong form is that the long axis is parallel with the vessel supplying the area affected. This is readily explained by the fact that bacteria travel most readily along natural channels. This tendency is noted most strikingly in miliary tuberculosis of the pleura. Large nodules result when there is a limitation of such extension by a process of beginning fibrosis. The larger nodules represent therefore an older and generally a more benign process.

The abdominal surgeon is able to distinguish two general groups on the basis of the size of the lesion. The very fine lesion, best designated submiliary, is seen in the more acute cases and the larger one, the miliary, is commonly seen in the usual case of slowly developing disease. Not infrequently the two are associated. This is evidence that an acute exacerbation has complicated the more slowly developing process.

The submiliary lesions are sometimes superficially situated and appear as though they might be scooped off without injuring the peritoneum. This type results from the development of the lesion

on the surface of the peritoneum and not in the subperitoneal vessels.

The tendency of all tuberculous lesions is to undergo caseation, as Virchow first pointed out. When this occurs, they lose their translucency and become opaque or cheesy. When this occurs the general contour is not changed, for, the center being dead, there are no further changes. In this it differs, it may be here remarked, from cancerous nodules. These, continuing the process of proliferation with subsequent contraction, produce a dimpling or umbilication of their summit. These, also being derived from a single focus, retain a spheroidal outline. The outline of a tubercle may be obliterated by the recurrence of secondary reactive processes at its periphery, producing an increased vascularization of its periphery. In hyperacute lesions, the general appearance of the peritoneum may be one of acute hyperemia with edema. Only on closer inspection can the fine tubercles be made out. The sense of touch may emphasize the ocular impression. If this vascularization produces a sufficient defensive reaction, the entire tubercle may be changed into connective tissue and the lesion healed. If, on the other hand, the defensive forces are inadequate, new tubercles may develop in the reactive zone. These, becoming confluent, encompass a zone so large that the nutrition of the center reaches so low an ebb that necrosis occurs. This necrosis differs from the caseation within the lesion, as will be noted below. This necrosis is due to vascular disturbances. When such a condition occurs destruction of tissue may be rapid and extensive.

The tubercle bacillus is capable also of producing a more diffuse infiltration without the production of definitely circumscribed lesions. This is particularly liable to take place in the subperitoneal tissue. I have seen a gut thickened to a centimeter for long distances without the macroscopic appearance of tubercles. It occurs, apparently, when the virulence of the infection is mild. At any rate, it occurs most extensively in animals when the inoculating material is heated to 60° before being injected into the animal. These lesions have nothing to distinguish them from other hyperplastic processes other than the demonstration of the tubercle bacillus. I have seen several specimens which were suspected of be-

ing tuberculous but definite proof could not be produced because of the failure to demonstrate tubercle bacilli.

*Microscopic Appearance.*—In artificially produced tuberculous lesions one is enabled to study to the greatest advantage the topographic relations of the disease in its various stages. The structure which enables us to determine by the topography the primary source of the infection is the membrana limitans. In brief, it may be stated that when the bacteria reach the site of the lesion by diffusion over the surface of the peritoneum, the lesion lies above this membrane; if by way of the blood vessels or lymph vessels, it lies below it.

From Borchgrevink's description I take it that his early stage represents a growth above this membrane, while his later stage presents an origin below it. This seems true because when bacilli become deposited upon the surface of the peritoneum an exudate furnishes the pabulum for their development. Cells gain access to this exudate secondarily. In this manner a tubercle of considerable size may be developing, and yet be quite clear and transparent, like a pearl, as Borchgrevink says, with only slight evidence of tissue reaction. In the second variety in which the bacilli find a nidus in the lymph vessels or blood vessels, there is an early dilatation of the vessel and a more diffuse area of infiltration about the developing tubercle. Sectioned at this stage, the elevation above the surface will be found to be very slight, and the membrana limitans goes uninterruptedly over it. As the disease progresses, the lesion enlarges and extends more prominently over the surface, and the area of vascular reaction becomes greater.

It is to be noted that after a lesion has developed for some time the specificity of the membrana limitans to dyes is lost, and its location can no longer be traced. All the acid-staining tissues lose their specific reaction and take on a reaction approaching the fibrinous; or, adopting a term used in the discussion of the development of fibrous tissue, they are in a precollagenous state. Therefore, in order to make use of the membrana limitans as a means of determining the origin of the disease, only the earlier stages can be studied. This method is adapted to experimental rather than to clinical or postmortem study.

As observed in the peritoneum in the human subject, it seems



likely that the small fine protuberant tubercles are derived from bacilli which have been deposited upon the surface of the peritoneum. This seems true even in those cases in which the mother tubercles were derived from direct extension or from venous or lymphatic transport.

What relation, if any, the means of transportation bear to the type of lesion produced can not be stated. From the uniformity of structure of the lesions and their superficial character it seems quite possible that those characterized by purely exudative products into the peritoneal cavity are due to superficial dissemination, while the more indurative and localized processes are more apt to arise more deeply in the tissues. The chief basis for this argument must be found in animal experimentation.

In children in the early stages, when there is a diffuse dissemination of tubercles, the irritation produces a profuse exudation of fluid, and ascites is the result. Whether this shall remain a simple fluid accumulation, or whether a fibrinous exudate shall result, depends on the fibrin elements which the exudate contains, not on the duration of the disease. If these are slight, either from too limited an irritation or from the presence of too great toxicity, the fibrin can not form, and the adhesive type does not result. On the other hand, if excessive fibrin is deposited, adhesion results.

If the disease extends more deeply in the tissues and the toxicity is greater, caseation and direct destruction of the tissue follows. Whether the tissue is better able to defend itself against bacilli which lie above the membrana limitans than it is from those which lie below it, is a matter of speculation. Animal experimentation would indicate that it is. In animals, conclusions must be drawn from histologic study, for all forms of the disease are fatal, sooner or later. Tissue destruction is greater in areas in which the bacilli are injected beneath the peritoneum than in lesions arising from intraperitoneal injections, and therefore arising above the membrana limitans. It is possible, however, that the subperitoneal injections represent a more concentrated infection than takes place on the surface.

The adhesive type is the least virulent of all. Instances in which the site of origin of a tuberculous process lies deeply in the tissues are more apt to be attended by caseation than are the more super-

ficial lesions. Of course there are wide individual variations. These may be explained by lessened resistance of the individual or greater virulence of the bacteria.

In the caseous type where intestinal loops are joined together, the union is not a true fibrous one, but rather an agglutination produced by the coagulation of debris. On section, such material presents a homogeneous granular field, devoid of elements capable of specific reaction to dyes. These deposits between the loops of gut correspond more to the stage of caseous pneumonia, as observed in lung tuberculosis, than to the fibrinous type.

That combinations of various forms occur is readily understood when we remember that one type of process may take place about an original lesion, while quite another type takes place in some other part of the abdomen. This is well shown in primary pelvic tuberculosis in which the tube itself is caseous with fibrinous adhesions about it, and an abundant crop of newer tubercles occurs over the remainder of the peritoneum, giving rise to an exudate. The great omentum may be involved in a thick tumor mass below the transverse colon. This mass contains caseated areas with considerable increase of fibrous tissue. The remainder of the peritoneal surface may be covered more or less by discrete tubercles, to the presence of which the peritoneum has responded by an abundant exudate.

*Classification.*—An endless number of classifications has been published. All recognize an exudative and a dry form. Charcot recognized a miliary, ulcerous, and healing stage. Among the more comprehensive classifications may be mentioned Münstermann's. He recognizes, from an anatomic view, a subacute miliary form and a fibrous adhesive form. Galvini recognizes five types,—a seromembranous, a serogranular, a purulent, a fibrous, and a cheesy form. Fenwick recognizes an ascitic, a fibrous-adhesive and an ulceropurulent form. A number of writers have made further subdivisions. Roersch divides the ascitic type into the free and the encapsulated. Thomas recognizes a dry and a purulent ulcerous type, and Margarucci distinguishes between an exudative type with and without adhesions.

To the pathologist the entire disease represents the same process differing merely in small detail. Given the fact that the disease

exists, as is manifest by the presence of tubercles, the surgeon is compelled to recognize that there may be an exudate, that there may be adhesions which he may be required to sever or let alone, and finally there may be caseation, which may permit easy puncture of the gut if he does not exercise great care. This simple classification seems as good as any for the purpose of keeping clinical records. At the operating table he can have no classification. Each part of the diseased area must receive its own interpretation in terms of pathologic anatomy.

The pathologic types above mentioned are capable of endless variation as to extent, rate of onset, and tendency to heal, dependent, not only on the character of the process *per se*, but also on the resistance of the individual, due to, or influenced by, inherent or environmental conditions.

*The Miliary Type, Submiliary Stage.*—(Fig. 196). In the earlier stages three characteristics may be recognized: superficial location, small size, and the absence of reaction in the surrounding peritoneum. These lesions may be compared with the point of a pin, if the ordinary tubercle is compared with the head. They are more nearly spherical, smoother, and more glistening than the ordinary tubercle. They are faintly translucent, and may have a slightly bluish shade. They have a striking resemblance to a small pearl. They may be more readily removed from the peritoneum than an ordinary tubercle, and they cause no hemorrhage when removed.

Weigert has emphasized characteristics of the lesions in cases in which the bacilli are strewn into the free peritoneal cavity from a primary focus. He believes that the peristaltic movements tend to distribute the bacilli throughout the peritoneal cavity; therefore the most numerous lesions exist in the places sheltered from such movements. Experiments with lampblack tend to substantiate this assumption. As already noted in the chapter on physiology, lampblack particles introduced into the abdominal cavity collect in the recesses beyond the reach of peristaltic movements. The distribution of tubercles is very similar.

This early stage is rarely observed in the human subject. It is only when primary lesions rupture into the peritoneal cavity that such a state can be observed. One sees them most frequently

in hernial sacs. In no instance should a clinical diagnosis of tuberculosis be hazarded if such are the only lesions present. This is particularly true when applied to the Fallopian tubes, and here often in response to some reactive process within the tube. These



Fig. 196.—Submiliary tuberculosis of the omentum. The gastrocolic omentum is thickly studded and the great omentum forms a solid mass along the lower border of the transverse colon.

papules, so far as I have been able to determine, are due to an exudate beneath the membrana limitans which coagulates and later becomes infiltrated with endothelioid cells with the subsequent formation of fibrous tissue. But at this point the process remains stationary.

**Miliary Stage.**—(Fig. 197.) As compared with the preceding stage the tubercles have become larger. In proportion to their size they project less markedly from the surface, and are united to the serosa by a broader base. The surrounding peritoneum shows an inflammatory reaction, the degree of which depends upon



Fig. 197.—Diffuse miliary tuberculosis of the peritoneum. These tubercles, contrasted with those in the preceding picture, are much larger.

how closely the lesions are situated together and perhaps upon the virulence of the organism and the ability of the tissues to react. The more deeply situated lesions produce more reaction than the superficial; or, perhaps, a greater reaction tends to cover the tu-



Fig. 198.—Fibrinous tuberclosis of the peritoneum. The long strands were formed by gently pulling apart coils of intestine which had become agglutinated.

bercles the less they project from the surface. If the reaction is great, the peritoneal vessels become prominent, the service vessels dilate, and the potential vessels quickly spring into prominence.

There may be considerable proliferation in the subserosa with a corresponding lessening of the mobility of the underlying organ. This thickening may be so great as to make the organ they cover palpable to the examining finger.

In the purely miliary form there is but little exudate, yet it is rarely absent. Usually if the fluid is not demonstrable clinically the case may be classed as purely miliary.

An exudate may form over the surface of the tubercle. When this organizes the tubercles are still less prominent, and they become less and less prominent as the exudate continues to form. If they are situated closely together, the membrane becomes thickened, often to an astonishing degree. When organization goes on along with this process, enormous hypertrophies may result. Caseation and hypertrophy may take place concomitantly in various regions of the surface.

*Fibrinous Type.*—(Fig. 198.) The general anatomic picture of this class may resemble the preceding closely, there being but the addition of a more or less abundant serous exudate. The character of this exudate varies in the different cases. If the fluid exuded in response to the tissue irritation remains uninfluenced by secondary factors, fibrinous bundles may be deposited and membranes and adhesions form. When the fluid remains in a free state, it is usually because it is too poor in fibrin, being more closely related to a transudate than to a productive infiltration. If there is great tissue reaction fibrin forms and the adhesive form develops. As a matter of fact there is usually more or less adhesion formation. If great toxicity exists the exudate remains fluid because coagulation is prevented because of toxicity. If only a partial coagulation is possible, a granular fibrin fills the spaces between the coils of gut and a false adhesion results. In this type the fluid likely becomes absorbed and the dry type results. At any rate in the dry forms there are usually some areas in which there is still free exudate, and these appear to be the younger lesions.

The character of the fluid varies in appearance, as well as in chemical and microscopic characteristics. It is usually thin and fluid, often lemon-yellow and sometimes greenish in color. Fibrin flakes may be seen. After being withdrawn it may either remain

fluid or coagulate spontaneously. In the latter instance a large fibrin mass often forms. Sometimes the entire mass solidifies. In some instances it may be jelly-like while in the abdomen. This is likely to occur where a small amount of fluid becomes encapsulated while the remainder of the abdomen is free from disease. The centrifugal specimen shows red blood cells, leucocytes, detritus, and some epithelial-like cells with granular protoplasm. Von Bauer states that the exudate is greenish or cloudy, or contains more or less blood. It is much less often bloody than in carcinomata.

The specific gravity at 18° C. is from 1.019 to 1.026. In one of my cases, a child of three, it was 1.015 to 1.016 at body temperature. The albumin content is 4.17 to 7.37 per cent.

Maurange's opinion that tubercle bacilli can always be demonstrated in the exudate has not been confirmed. Even in animal experimentation most guinea pigs remain negative when injected with peritoneal exudate. Wyssokowicz is of the opinion that guinea pigs can always be inoculated if enough of the exudate is used. It is said the skin of a black cat will cure whooping cough if the skin is black enough.

The amount of fluid varies greatly. It may be so slight as to escape detection during clinical examination, and its presence be unsuspected until the abdomen is opened. This is particularly likely to be the case in localized forms, particularly those involving the Fallopian tubes only or those confined to hernial sacs. On the other hand, the amount of fluid may be enormous, being rivaled only by the exudates associated with adhesive pericarditis. In such instances the life of the patient may be menaced by the increased intraabdominal pressure.

Tuberculosis of the peritoneum may imitate all other diseases that occur in the abdominal cavity. Dropsy, cysts, inflammation, adhesion, tumors, intestinal obstruction, abscess, fistula, all come within its repertoire.

*The Adhesive Type.*—(Fig. 199.) As previously noted, the question of the formation of adhesions is one of the degree and character of fibrin content in the exudate, and not a question of stages or duration. It is only in the type characterized by reactive response that adhesions can occur, that is, a substance which is capa-



ble of forming fibrin must be exuded. This takes place only when the exudate is relatively slight in amount, just sufficient to fill in

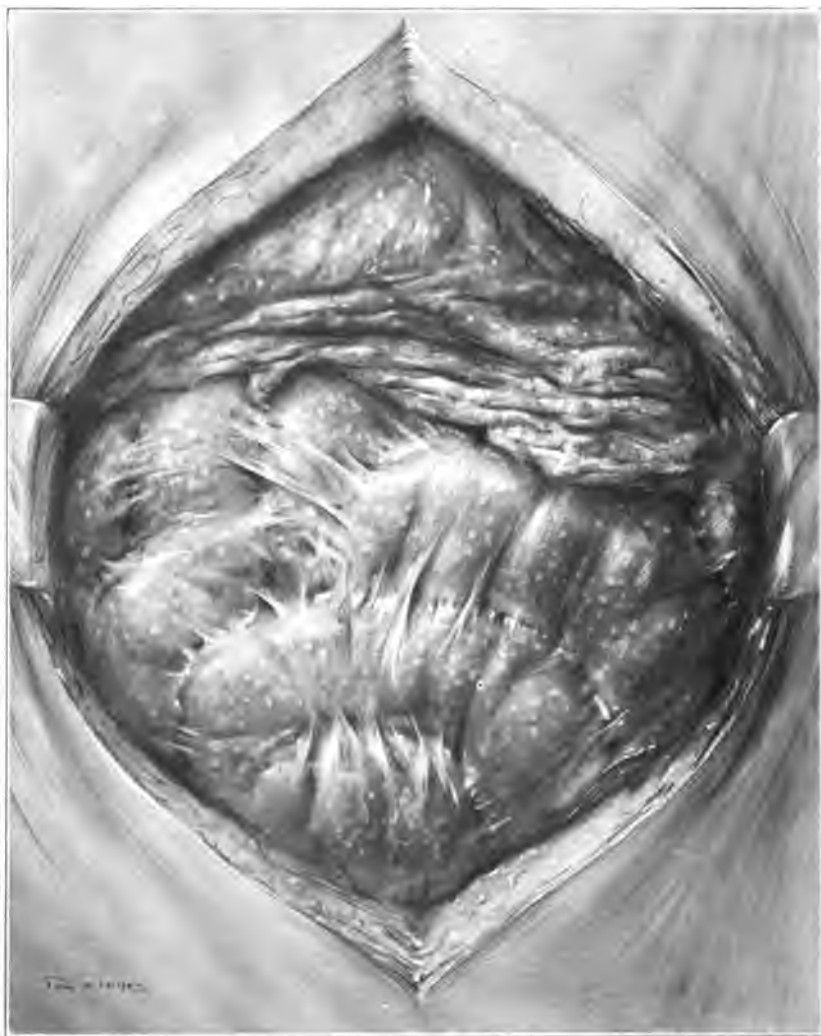


Fig. 199.—Adhesive caseous tuberculosis of the peritoneum. Contrasted with the preceding cut the fibrous bundles show a greater advancement toward organization, but even these resorb when the disease progresses to recovery.

the sulci between the intestinal layers. This exudate, coagulating, forms a fibrin which undergoes some of the changes incident to

the formation of fibrous tissue. It is rare, however, that fully developed fibrous tissue is formed. Usually the fibers, even in relatively old adhesions, still take the picric acid, and refuse the fuchsin, in Van Gieson's stain. The formation of adhesions merely means that the tissues are reacting and are capable of repairs. Adhesions are but an expression of this state: they are not an essential part of the healing process. Given the reaction in the gut wall, healing would take place if no adhesions were present. Usually, as the healing process progresses, the adhesions loosen and are absorbed.

The relation of adhesions to exudate and caseation is only one of association. Allport maintains that an exudate hinders healing. At any rate, the formation of an abundant exudate of a low specific gravity indicates that the reactive power of the tissue from which the exudate comes is very low or they have not been stimulated to full reaction. If exudate is present in such an amount as to interfere with peristalsis and circulation, it is no doubt capable of exerting an influence inimical to healing, and its removal may aid in restoring equilibrium. The productive activities may continue operative in the subperitoneal tissue. There then results a great thickening of the walls. This is particularly apt to take place when the primary area of infection is limited, and it is noted most often in the region of the appendix and cecum. Here the enlargement may be very marked without there being surface lesions of tuberculosis. In many of these microscopic examination alone can determine the tuberculous nature of the lesion.

*The Caseous Type.*—(Fig. 200.) When the destructive processes continue, the tissue is destroyed. This process is the same here as elsewhere. Toxins cause a liquefaction of the tissues, and an ulceration results. Usually the ulcerative stage has been preceded by a less destructive process, and adhesions have already formed. The two degrees of involvement may be playing their respective parts at contiguous or more remote regions of the gut. In this way adhesions and ulceration may coexist. With ulceration an exudate that is converted into a granular substance may form. This, in a measure, agglutinates adjoining coils of intestine, and protects the environment somewhat should perforation take place. Usually, the same sort of material has collected in the interstices of the

peritoneum, causing a thickened gut. This thickening is not comparable with the increase in the connective tissue in cases characterized by marked reaction. When such ulcerated areas heal, it is distinctly by secondary intention. The affected areas must be pervaded by materials capable of fibrin formation. This is possible only after the virulence of the infection has somewhat abated.

*Location.*—The favorite locations for the tubercle eruption are the cavities about the liver and spleen, the surface of the mesentery, and in the pelvis. There can be but little doubt that gravitation plays an important part in the dissemination of the bacilli. Experimentation and clinical observation both bespeak this fact.

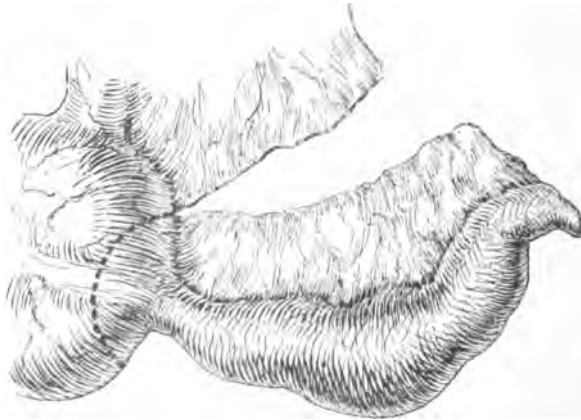


Fig. 200.—Caseous tuberculosis of the appendix. The external appearance showed only uniform enlargement. On section the walls showed many areas of advanced caseation.

The anterior abdominal wall, being protected by the great omentum from the sweeping action of the intestine in peristalsis, is often the seat of conglomerations of tubercles. The great omentum too is exposed to infection. If the process in the omentum becomes extensive, adhesions with the abdominal wall are prone to take place, or, as is uncommon, it rolls up beneath the transverse colon. The position of the omentum may be an index to the site of origin.

*The Relation of Cirrhosis of the Liver to Tuberculosis of the Peritoneum.*—As already noted, the liver is found to be cirrhotic in a large number of cases of peritoneal tuberculosis. Cummins

found this to be the case 10 times in 82 cases; Münstermann, 6 in 46 cases; Friedländer, 5 in 88; Nothnagel, 2 in 13; Heintze, 6 in 28; and Vierordt, 5 in 25, which, collectively, is about 12 per cent of liver involvements. This frequent association indicates a causal relationship. Whether the stagnation of the abdominal circulation forms a favorable nidus for the infection (Weigert, Wagner, Grawitz), or whether the liver is secondarily involved from the peritoneum, is not certain. The weight of opinion seems to favor the latter assumption. Cases in which the pericardium is involved, producing a hyperemia of the liver, are thought to be particularly predisposed to liver sclerosis. Birner holds this view, as do also Grawitz, Weigert, and Rokitsansky.

Woolley (quoted by Rolleston) collected 90 cases of cirrhosis of the liver in patients under twenty-one. Ascites was present in 59; and in 12 of these tuberculous peritonitis was diagnosed.

*Tuberculous Cysts.*—Unique in the pathology of tuberculous peritonitis is the case of a cyst reported by Geipel. The patient was a woman, age twenty-six, who had died of pulmonary tuberculosis. Autopsy showed tuberculosis of the gut tract, but no ascites. At the free border of the ileum five cysts were found. Microscopic examination showed the cysts to be formed between lamellæ of the hypertrophied peritoneum in the region of the tuberculous ulcers of the gut. The cyst contents consisted of a granular, coagulated mass. The author regards the cyst as having been formed in lymph vessels, the outlet of which was constricted by the fibrous tissue in the ulcer. He believes that the cyst wall proliferated in order to keep pace with the developing cyst.

It has never been my fortune to observe a cyst of the magnitude reported, but I have seen several very small ones. It appeared that these were formed extraperitoneally, independent of preexisting lymph spaces. I have observed them most often on gonorrheal pus tubes. Here they may resemble small, clear tubercles and are no doubt often so diagnosticated. My opinion is that they belong to the encysted spaces above mentioned. The author above mentioned noted that the weakness of his theory is found in the varying thickness of the cyst wall, a fact which would speak strongly for the theory I have advanced.

As a corollary to this is the vastly more common so-called

encysted tuberculosis. This condition is brought about by the adhesion of neighboring structures imprisoning a certain amount of exudate. The condition is very common when the exudative and fibrinous forms coexist.

*Bovine Tuberculosis.*—The relation of human to bovine tuberculosis has not been fully established. The occurrence of the bovine type in the human subject has not been established with certainty. Nevertheless, cases which resemble in their anatomic form the type seen in cattle are occasionally met with in the human subject. An attempt will be made here to present an outline based on the cases recorded, without an attempt to discuss the question of etiology.

Virchow reported the first case recorded, under the impression that it was a type of sarcoma. Bizzozero reported the first case correctly diagnosed. He emphasized the fact that pedunculization is the characteristic factor.

Creighton reports 12 cases, in four of which, however, the peritoneum was not affected. Jurgens described a case in which the lesser omentum was the chief seat of trouble, though there was thickening of the pleura and pericardium. The most perfect example recorded is that of Ipsen. The omentum was adherent to the abdominal wall, and the sigmoid and small intestines were agglutinated. There were many tubercles over the peritoneum of both the small and large intestines. Some of these tubercles were flat, others sessilated, and others pedunculated. There was no free fluid. The retroperitoneal lymph glands were large and cheesy.

It is the large size of the tubercles, and particularly the disposition to form a restricted base, that is characteristic. The large lesions may develop from a single center, or several isolated centers may become confluent, producing a large lesion. Thus Uffenheimer reports a case in a child of one year in which lesions the size of a nickel were observed. MacCallum records a case in which the individual lesions varied from 2 mm. to 2 cm. in size. Some of the nodules were imbedded, but others hung free. Some of them hung by stalks up to 10 or 12 cm. in length. All of these pedicles contained fluid.

The importance of the bovine type from a clinical point of view is that its lesions are copied by metastases from a papillary

cystadenoma of the ovary, even to the grayish or reddish color. According to Orth this form is sometimes seen in the human subject. According to Troja and Tangl it is possible to so attenuate the human bacillus by means of iodoform that lesions resembling the bovine type may be produced in rabbits.

*Healing Process.*—Bumm sums up the healing process as follows: (1) cell infiltration of the tubercle and its environment; (2) degeneration of the giant cells and epithelioid elements of the tubercles; (3) increase of surrounding connective tissue and encapsulation of the tubercles; (4) formation of a scar nodule. Gatti, Nannotti, and Baciocchi came to the same conclusions from experimental study. Hermann has recently repeated these experiments, and illustrated his paper with drawings of his work. According to him about forty days are required in rabbits before vascularization and fibrous tissue formation is at all advanced. That tubercles experimentally produced may become vascularized and inclosed by fibrous tissue is no doubt true; but this does not assure a cure of the disease, for at this time the lesion contains viable bacilli, as may be proved by inoculation into fresh animals.

The disposition to the formation of fibrous tissue is likewise evidenced by the formation of pseudomembranes. Here, however, as about the tubercles, the fibers are precollagenous, and do not form a barrier to the spread of the infection.

Gatti, on the contrary, did not find healing by the formation of fibrous tissue, but by hydropic degeneration, first of the cell protoplasm and then of the nuclei of the endothelioid cells.

Generally speaking, the process of healing is much the same as that noted under the chapter on the formation of adhesions. Under favorable conditions fibrous tissue is formed. The major part of the exudate is absorbed without the formation of demonstrable scar. The foreign material is removed by phagocytosis, and the peritoneum may be left as smooth as formerly.

*Formation of Fistulas.*—Brichet was the first to describe a case of tuberculous peritonitis in which pus escaped from the umbilicus, but the first adequately described was by Dresch. Previous to this Goebel had collected four cases. In the same year Gauderon reported a case with recovery. A complete review of the literature was made by Ziehl. He presented abstracts of 30 cases. Of his

cases 20 were in children and 10 in adults. Of the 20 cases in children the opening lay in the umbilicus in 18. In 5 there was a direct communication between the gut and the abdominal wall. This abscess then perforated the abdominal wall. The extent of the opening varied. In 3 permanently, and in 2 intermittently, all the feces escaped by the artificial opening, presenting, therefore, fecal fistulas. Of the 10 adult cases, 5 perforated in the umbilicus. In two the gut was directly attached to the abdominal wall. Very recently Cullen presented an excellent review, together with a presentation of the case histories of 19 cases.

The pathogenesis of these perforations may be as follows: the gut may become adherent to the abdominal wall and the destructive process gradually perforates, producing a direct connection between the gut lumen and the external world. On the other hand, the opening in the gut may communicate first with a walled-off space within the peritoneal cavity, a secondary ulcerative process being necessary to perforate the abdominal wall, as in Borchgrevink's case. Obviously, in the latter instance perforation into the peritoneal cavity is more liable to occur and lead to a fatal general peritonitis.

The reason for the more frequent perforation at the umbilicus is, obviously, the fact that this is the thinnest point in the abdominal wall. Possibly the round ligament or the urachus may act as a gubernaculum for the conduct of the process. The absence of a muscular layer, a tissue always unfriendly to the tuberculous process, furnishes an additional reason why ulceration extends to this point. If distention of the abdomen has preceded, or accompanies, the tuberculous ulceration, the scar closing the umbilical opening may become much attenuated, even to the point of bulging. Congenital weakness may contribute to the ease with which perforation may take place. Of more significance than this would be the presence of an open ductus omphalomesentericus or a Meckel's diverticulum, along which the infection might extend (MacSwiney).

In the majority of cases a mixed infection precedes the perforation. Thus Bertherand opened what he thought to be a skin abscess. At the autopsy the pus cavity was found to be surrounded by tuberculous masses, and communicated with the transverse co-

lon. It is possible that in some cases a peritoneal abscess perforates the abdominal wall, and, the pressure being relieved, the intestines perforate into the abscess. At least that would be my interpretation of Knecht's case.

Voigt reports a case in which an umbilical hernia developed rapidly during the early stages of a peritoneal tuberculosis. If, as in this case, an omental adhesion forms, the most favorable conditions are provided for the perforation of the abdominal wall at this point.

A consideration of the pathogenesis permits us to deduce that the prognosis is very grave. One-third die within ten days after the perforation takes place. If death is deferred, the prognosis is then dependent upon the extent of the underlying process and the effect the fistula may have upon the nutrition of the patient.

When such a fistula exists, a prolapse of the gut mucosa may take place through the fistula. Pels-Leusden reports such a case, occurring in a child aged three, which followed laparotomy for the cure of tuberculous peritonitis. The condition was relieved by incising the skin about the opening, and folding the edges together, thus permitting the closure of the opening.

Maurange states that perforation of one intestinal loop into another sometimes takes place. I have been unable to find a specific instance. The same may be said for perforations into the vagina and uterus.

Communications between the gut and the bladder are, fortunately, of rare occurrence. Ulzmann and Schütz each report a case.

Among the rare perforations, fortunately, is that into the blood vessels. DeMussy and Trabaud each mention such a case. Embolism and hemorrhage must be the end in such cases.

*Perforation into the Peritoneal Cavity.*—Instead of perforating through the abdominal wall with the formation of a fecal fistula the perforation may occur into the general peritoneal cavity or into as much of the cavity as may not be walled off by adhesions. If the site of perforation points into a pocket formed by the adhesion of the intestine and omentum a fistula may result by perforation of the abdominal wall, as previously described. If there are no adhesions present, a general peritonitis is the result. This is the type now under consideration. The cases reported in the liter-



ature are not numerous. It is reported that Louis XIII died of such a condition (Berard and Patel).

In several of the reported cases it is not clear whether a tuberculous ulcer of the gut or a peritoneal tuberculosis was the cause of rupture. Thus Pasquet reports cases of perforation in patients afflicted with pulmonary tuberculosis. Some of the cases published under this head were clearly ulcers of the gut. Paulicki reported a case belonging to this category. Cases reported by Labbi and Beale probably belong to this class. The cases reported by Letulle were probably cases of perforation in tuberculous peritonitis. Oppenheim and Lanbry report two undoubted cases, and Lodure reports three. The perforation ranges in size from 3 mm. to 4 mm. in diameter to an opening involving half the circumference of the bowel (Labbi). The perforations are usually solitary, but as many as five have been reported. The most frequent site is in the terminal ileum or at the ileocecal junction, but any region may be involved. Darrier observed one near the duodenum. Corbin has reported perforations of the appendix, and Simon and Chatin have reported perforations of the large gut.

The accident is rare. This can be explained by the fact that peritoneal tuberculosis is primarily a productive inflammation, and the disposition to form adhesions precludes the probability of perforation. Statistics bearing on this subject for the most part antedate the period of exact diagnosis in abdominal diseases. Eigenstedt in 566 autopsies on the tuberculous saw perforations 26 times. The existence of a stenosis predisposes to the formation of ulcer above the stenosis and a subsequent perforation.

When perforation occurs in the absence of adhesions a diffuse peritonitis develops. Because of the abnormal state of the peritoneum, a protective reaction of any great degree is not possible. The symptoms and termination are those of an acute generalized peritonitis.

**Symptoms.**—In no other disease is the onset more variable and there is scarcely a disease of the abdomen that can not be mimicked by tuberculous peritonitis. Errors of diagnosis arise most frequently from failure to recognize its protean character and are more common in regions where the disease is relatively uncommon.

The onset may be sudden as a perforation, brusque, even brutal,

as Dupre and Ribierre express it, or prodromal symptoms may extend over many years. These two extremes may be blended by general ill health with exacerbations. Accordingly it is advantageous to separate the acute and chronic types.

*Acute Type.*—Rolleston says the onset is acute in one-third of the cases. Stone, Bottomly and Shattuck emphasize the importance of the acute cases. According to these authors ascites in children developing suddenly is usually due to tuberculous peritonitis. The acute cases begin either as an acute infectious disease or as an abdominal crisis. Chill, fever, headache, vertigo, and malaise characterize the former type, while vomiting, distention and pain mark the onset of the latter. The conditions may be combined. When fever and malaise predominate, typhoid fever may be simulated. This resemblance is heightened by distention and general abdominal tenderness and pain.

The acute, so-called typhoid form, is very rare. Reports of six cases are presented by Vierordt; one by Kyburz, one by Fenwick and one by Schmallfuss. The chief symptom is the continuous high fever, even to  $105^{\circ}$ , often with bronchitis and vomiting, sometimes with roseola exanthema and swelling of the spleen. In all there was an intense tuberculosis with thick pseudomembranes and adhesions, with but slight affection of other organs. In two of Vierordt's cases the exudate was serous-hemorrhagic. It is not uncommon for such cases to simulate acute perforative conditions. Thus Halstead reports a case which began with chill and temperature of  $103^{\circ}$  and severe abdominal pain which localized in the region of the appendix. Bonet reports a similar case. Not all cases of this type begin so brusquely, however. A case with rapidly developing malaise may present no other symptom than an increase of the girth. Bladder irritation may be the first symptom. A hernia, long existent, may become more prominent and feel uncomfortable.

When the peritoneal affection is only a part of a generalized miliary tuberculosis, the peritoneal involvement may be overshadowed by the severity of the general infection. Conversely, the general infection may be dominated by the peritoneal involvement. For instance, in a patient I once observed, following an injudicious local resection of an epididymal tuberculosis, an acute abdominal

crisis developed. The autopsy disclosed an almost universal miliary involvement without any apparent reason for the predominance of abdominal symptoms.

Generally, however, the location of the predominant infection may be determined from the symptoms. Pleural and pulmonary involvement are recognized by the pain, exudate and respiratory disturbance; cerebrospinal, by the characteristic symptoms. However, the involvement of the abdominal surface of the diaphragm may produce a rapid respiration, an extensive exudate may cause dyspnea from pressure and a primary cerebrospinal involvement may be characterized by early abdominal distention or retraction and vomiting.

Unfortunately the differentiation of the predominant location of these acute generalized infections is of academic interest only, for the result in each is the same. Yet, if it be recognized that the abdominal lesion is but a part of a generalized process much embarrassment may be spared the surgeon.

Not infrequently an acute onset may involve a limited region of the peritoneum only. The most frequent sites are the Fallopian tubes in the female and the ileocecal region in the male. In the former situation an acute salpingitis may be simulated and in the latter an appendicitis. This latter type was first described by Lejars; since by Guillemare and Rousseau. In this type the pain may be sudden in its onset and confined to McBurney's point.

The first symptom may be the sudden appearance of hernia or pronounced irritation in a previously existent one. The distention, due to the exudate, may open a patent sac. Operation for the cure of the hernia may reveal the peritoneal tuberculosis (Rolleston and Wright).

Because of the rarity of the acute lesion in comparison with the diseases it simulates, the diagnosis is usually made at operation. It is only when there is obvious tuberculous disease elsewhere that one is led to suspect a like condition in the local lesion.

*Chronic.*—The chronic type is characterized by an indefinite onset. Prodromal symptoms may exist for months or years. Lauper's cases varied from 18 months to 10 years with 50 per cent lasting more than a year. There may be malaise, general weakness, indefinite pain in the abdomen and possibly intestinal dis-

turbance. Local symptoms may predominate. Dysmenorrhea, sacral pains, leucorrhea or referred pains common to irritation of the pelvic organs may be present when the pelvic peritoneum is involved. Periodic or persistent constipation with localized pain in some region, notably in the region of the umbilicus, less often in the region of the ileocecal valve or transverse colon, may present the first symptoms of the disease. A neoplasm may be simulated, because of tumor, pain, obstruction and exudation. Fecal impaction caused by overstretching of the part may simulate a tumor.

Classification of the clinical forms of chronic peritoneal tuberculosis presents difficulties equal to those already encountered in the discussion of the pathology. Nevertheless, here as there, a certain advantage comes of the consideration of the dominant types. There is an advantage in adopting here the same classification followed in the discussion of the pathologic anatomy.

*Miliary Type.*—In most of the cases where there is a sudden onset, the disease presents the miliary stage. The same is true of the localized lesions when pain is a dominant factor. In some of the hyperacute types a preliminary stage may be said to exist since the general symptoms of inflammation are predominant with few or no tubercles.

On the other hand tubercles may be found in the course of operations for conditions in which it is not expected. Just how frequently this occurs can not be determined from the literature, because in most cases the diagnosis depended on clinical observation alone. A study of my own material shows that the most of the cases presenting patches of small tubercle-like nodules in the neighborhood of chronic irritations were not tuberculous at all. The clinical diagnosis in such cases is not reliable.

From the foregoing it will be apparent that this type of the disease is the least characteristic of all. Sudden stormy onset on the one hand, and vague indefinite pains on the other, is not sufficient to warrant any diagnosis, and if the surgeon is confronted by such a condition his first thought should be not chagrin, but a curiosity to determine by microscopic examination whether or not it is really tuberculosis.

*Fibrinous Type.*—In this type, usually following some of the prodromal symptoms, fluid is present in the peritoneal cavity. The

increase in girth may be the first sign that convinces the patient that he is ill. This phenomenon usually excites the apprehension of the most plethoric patient. Sometimes he expresses surprise that, notwithstanding the progressive weakness, he is gaining in weight. In nearly every exudative case the anamnesis will bear record of some prodromal symptoms. Rolleston notes that in children the intestines may be so filled with fluid that a pseudofluctuation is imparted to the examining hand, which may be mistaken for ascites. The outline of the abdomen is characteristic particularly in children. There is a general abdominal enlargement (Fig. 201) involving all regions of the abdomen alike. The more acute the disease, the more marked is this fact.

Fluid is naturally detected in the flanks first. In purely serous cases it will change its level with change of position. In extreme



Fig. 201.—Outline of extreme abdominal distention in a young girl with miliary tuberculosis of the peritoneum.

cases nearly the entire abdomen may be filled with fluid. When the process is at all extensive and adhesions are not present, the characteristic signs of free fluid in the abdomen may be obtained. It has seemed to me that in tuberculous peritonitis the wave obtained by tapping is less distinct than in other exudates of like magnitude, and that when the position of the patient is changed, the line of the upper layer of the fluid changes less promptly than in other affections attended by exudation. Attention to these points has enabled me in several instances to correctly surmise the tuberculous nature of the disease where sarcomatosis had previously been diagnosticated. Bulging in the pouch of Douglas may be made out by vaginal palpation. Thomayer believes that the presence of ascites predominantly in the left flank is particularly

characteristic of exudates due to tuberculosis. He explains this by assuming that, when the great omentum retracts, it draws the intestines with it, leaving the fluid to occupy the left portion of the abdominal cavity. Numerous observers have confirmed this sign.

After a puncture, in some instances, a rubbing of the roughened peritoneal surfaces upon each other can be heard or palpated during respiration. If distention is not too great, this sign may be elicited without removal of any of the fluid, particularly if the patient can be taught to exhale suddenly.

In some instances the amount of fluid is not great, but may be as much as two to six gallons. In rare instances in which acute exacerbations occur, the pressure from the fluid may be so great as to endanger life. In one of my patients 16 liters were removed.

*Adhesive Type.*—This type may be associated with or follow the ascitic or may occur independently. Its general onset presents much the same sequence of symptoms. When coils of intestines become adherent to each other and to the omentum, pockets may form which become filled with fluid and present the physical characteristics of cysts. These cysts are particularly apt to form in the upper abdomen. The thickened gut may present palpatory evidence of a solid tumor. On vaginal examination the thickened tube may have anchored the uterus and the bulging fluid from above may present a sacculated semifluid resistance on either side of the tubal ridge. Disturbances due to lessened intestinal motility often occur. The great omentum may be much thickened and attached to coils of adherent gut, or it may form a tumor of itself.

*Caseous (Ulcerous) Type.*—In this type there is no free fluid, but there are bossilated masses and great disturbances of intestinal mobility. If the process ulcerates through the intestinal wall, as not infrequently happens, a mixed infection takes place in the retroperitoneal spaces. The uterus is nearly always fixed, and usually the omentum, much thickened and rolled upon itself, presents somewhere in the abdomen as a palpable tumor. The surfaces of the parenchymatous organs, liver, spleen, etc., are apt to be infiltrated, complicating the picture.

Digestive disturbances are at their height in this type. Pain, meteorism, and diarrhea occur. Fever increases as the disease progresses. The umbilicus may be distended, everted and reddened,

showing distended capillaries near its border and radiating veins extending over the contiguous skin. This sign is of great value when present, but it is rare. Thus Heintze noted it twice in 25 cases, and Häne not at all in 46 cases.

*Localized Form.*—Frequently the process is localized in one region of the abdominal cavity. The symptoms may be local, but the process diffuse. For instance, a chronic process may exist about the cecum or tubes, while the whole peritoneal surface is sprinkled with a younger crop of tubercles. The conditions here considered are those in which the whole process is played in a localized region of the abdomen.

*Ileocecal Tuberculous Peritonitis.*—Whether or not tuberculous peritonitis is primary in this region is a matter of opinion. Conrath believes it is rarely so in the adult. Hartmann and Baum believe it is frequently primary. The latter out of seven cases found no other lesion in four. Most convincing are the statistics of Campiche. He collected 279 cases, in which more than one-half were apparently free from the disease elsewhere. Weiner makes the point that the existence of a slight pulmonary tuberculosis does not demonstrate that the cecal lesion is secondary. From the clinical point of view the important point is whether the lesion is local or diffuse within the abdomen.

The general opinion is that the disease begins in the mucosa and extends to the peritoneum. This must be true for most cases because a true tuberculoma, palpable through the abdominal wall, is often produced.

Ileocecal tuberculosis manifests itself either as an acute process, simulating acute infection by pus organisms (Fig. 202), or as a chronic fibrosing process simulating a neoplasm.

The former type is often mistaken for appendicitis and operated on as such. The acute onset with mass formation accounts for the confusion. Andrews suggests as differentiating points the greater density and greater motility of the tuberculous lesion. Following this suggestion I made such a diagnosis in a young woman who had a hard tumor in the ileocecal point which was freely movable except downward. At operation a mass of indurated omentum was found wrapped about the distal end of a long appendix which harbored an enterolith which had given rise to a

suppurative periappendicitis. Nevertheless, Andrews' point is well conceived, and should work out as a rule. Localized tuberculosis of the appendix is occasionally met with. It may be ulcerous, giving rise to infection of the lymph glands of that region. The chronic hypoplastic form is more common. In scrofulous persons

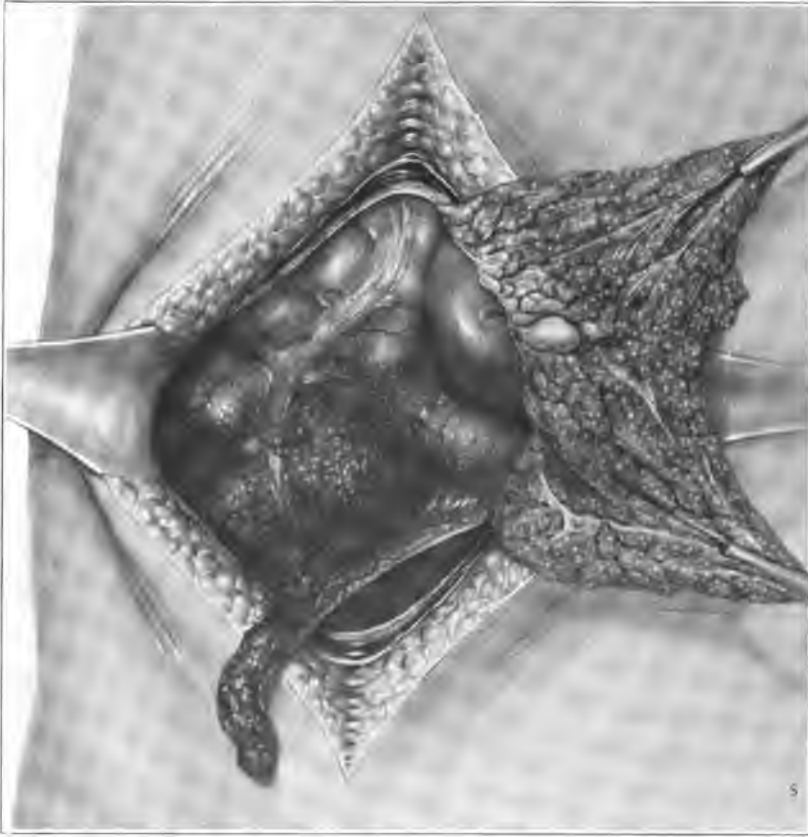


Fig. 202.—Acute miliary tuberculosis of the ileocecal region, young man aged 19. The great omentum is being drawn out of the wound. Save for a number of mesenteric lymph glands, the entire disease is exposed to view.

with subacute inflammation in the appendicular region a tuberculous lesion may be suspected, particularly if a mass is palpable. This type must be differentiated from carcinoma. The presence of tubercles in other regions of the peritoneum is often the best dif-



ferentiating sign, though one may conceive of the possibility of a miliary carcinosis producing a similar picture.

The only other disease which could be confused with tuberculosis of this region is actinomycosis. The differentiation likely must be made at operation. Tendency to invade the retrocecal tissue may suggest the possibility of actinomycosis (Teckener). It is only the presence of the canary bodies that make a certain diagnosis possible at the operating table.

This type resembles woody phlegmon of the neck. These conditions are difficult enough to differentiate from hyperplastic tuberculosis in the laboratory.

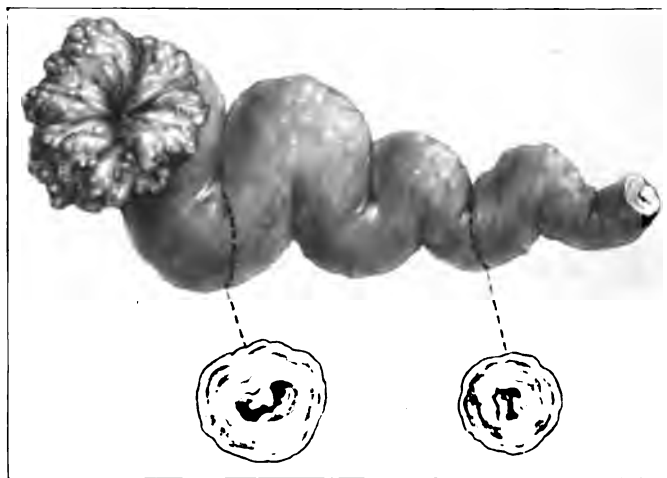


Fig. 203.—Primary peritoneal tuberculosis of the Fallopian tube. Except for a few tubercles on the pelvis peritoneum, no other lesion except in the tubes was to be seen.

On the whole the indurative diseases in the ileocecal region are difficult to differentiate. Hill made a statement to the effect that no one should undertake an operation for appendicitis unless he is capable of resecting the cecum, should conditions demand it. This is certainly true when tuberculosis or carcinoma is a possibility.

Therefore when there is a hard, more or less movable mass present the operator should anticipate the possible need for a resection of the cecum and the surgeon should be assured that the necessary skill and instruments are at hand to cope with any emergency.

The tumorous form is slow in onset and sometimes first gives evidence of its presence by producing a constriction of the lumen of the gut, simulating in this certain types of carcinoma.

*Tuberculosis of the Pelvic Peritoneum.*—Tuberculosis of the pelvic peritoneum is usually associated with involvement of the deeper structures even to the lumen of the tubes. If we regard tubal tuberculosis as primarily of two types as some authors do, endosalpingitis and perisalpingitis, it is the latter which is primarily peritoneal (Fig. 203). The equivalent of this classification is that commonly used, namely, the ascending and descending. The former classification is preferable since it implies pathologic relationship while the latter suggests the possible etiology. The etiologic relations of this lesion have been sufficiently considered and it remains only to discuss the pathologic features.

Peritoneal tuberculosis of the tubes may present any of the types already discussed. Typical primary miliary tuberculosis of the tubes is said to be uncommon. In fact Daurios denied its occurrence. I have observed several cases in which such condition existed. Many of the cases recorded seem to indicate that this type is common. Evidence is sometimes lacking which would separate these cases from pseudotuberculosis. Not uncommonly in septic infections of the peritoneum of the tubes flakes of fibrin become deposited on them. These organize, forming small granular elevations which resemble tubercles very closely. Sometimes a positive diagnosis is possible only after a careful microscopic examination. Williams is of the opinion that the lesion is usually primarily submucous approaching the peritoneum secondarily. This is substantiated by the fact that the common form is the caseous. Breaking down of tissue is apt to be early, and once the lesion approaches the surface early attachment to surrounding surfaces takes place. In this way, a mass is produced formed by matted adnexa and surrounding organs. Between these structures a granular material, formed from the exudate, is found. The matting may be so extensive that there may be no free surface presenting miliary tubercles.

The important clinical factor is the differential diagnosis. Very commonly where a chronic irritative process has existed for some time small vesicle-like nodules are formed which in a very super-

ficial way resemble tubercles (Fig. 204). These cysts are in structure identical with the stalked hydatids so commonly observed hanging from the fimbriated ends of the tubes. They are miliary cysts filled with a clear fluid. The walls of the cysts are composed of a very fine layer of connective tissue and are lined with a flat endothelium and covered by the same kind of cells. When these cysts are shrunk in alcohol these cells are nearly cubiform. They owe their origin to the deposition upon the surface of the peritoneum of any flocculent precipitate, the formation over these of a pseudoperitoneum which forms actual peritoneal cysts. Occasion-



Fig. 204.—Small subperitoneal cysts of the tube. These are often mistaken for tubercles. There is no infiltration about them and they stand above the surface of the peritoneum.

ally, particularly over the fundus of the uterus, these cysts may be as large as peas or even larger. The larger ones readily collapse when punctured, but the small ones can not be recognized. The lack of any reactive process about them, either vascular or indurative, and the semitransparent appearance are sufficient to differentiate them from tubercles. Sometimes there are small granular nodules which appear much like the cysts, but which are made up of granulation tissues covered by a new endothelial layer (Figs. 205 and 206). These are less transparent than the cysts and may



Fig. 205.—Granulomatous nodules of the tube and ovary in an old infected tube in a case of myoma of the uterus. The outline of the nodules is irregular, indicating growth by deposition rather than by expansion as in the case of tubercles.



Fig. 206.—Granulomatous nodules on a chronic pus tube and ovary. These are formed by the development of foreign body tubercles below the peritoneum and the deposition of fibrin over the area so irritated or by the organization of such deposits.



**Fig. 207.**—Foreign body giant cells from specimen shown in Fig. 205. Plasma cell infiltration causes these areas to resemble tubercles but the giant cells have central nuclei and the protoplasm has not undergone caseous degeneration.



**Fig. 208.**—Microscopic section of the specimen shown in Fig. 206, showing foreign body "tubercle" developing on the surface of the tube.

attain a size varying from a pinhead to a split pea. Occasionally foreign body giant cells are found in them (Figs. 207 and 208).

On the other hand a subperitoneal tuberculosis of so slight a degree may exist that its nature is not suspected until the tube is sectioned. In such tubes the involved area may be detected on palpation when not discernible by inspection. Williams has called attention to this type and has recorded several cases.

The indurative type, in which the subperitoneal tissue is extensively hypoplastic, may resemble gonorrheal salpingitis very closely. If caseated areas are discovered or if tubercles are seen about the borders of the process the tuberculous nature of the process may be recognized. If there are none such, perhaps a section of the tissue will show fine granulations suggestive of tuberculosis but miliary areas of necrosis are sometimes noted in gonorrheal tubes. In such borderland cases microscopical examination may be necessary before the nature of the process can be determined. Equally confusing are the chronic gonorrheal tubes with small cysts or granulations on their surface (Fig. 208).

A localized thickening of the tube may be caused by tuberculosis, presenting a veritable tuberculous salpingitis nodosa. The nature of these can usually be detected by section with a knife when caseated areas become apparent. These may resemble an ordinary salpingitis nodosa. It is just possible that an ordinary salpingitis nodosa may be due to healed tubercles.

It will be apparent from the foregoing that while generally speaking tubal peritonitis is easily recognized at the operating table, frequently cases will be encountered which must be followed to the laboratory before the diagnosis can be made.

Sometimes the bottom of the culdesac is studded with tubercles while the tubes are relatively free. This does not take place as an isolated disease but when other parts of the abdomen are involved the most pronounced lesion may be found here. The thickening may be so pronounced that it can be made out by vaginal palpation.

*Tuberculosis of the Hernial Sac.*—(Fig. 209). In a number of instances cases have been recorded in which the sac in hernias has been the chief or exclusive portion of the peritoneum involved. The first cases recorded were by Cruveilhier, followed many years after by the report of one case by Hayem. Collective papers have

been presented by Brunns and Haegler. Roth presents 22 cases, supposedly all that were reported to date. Finally Kohler collected 36 cases, and presented a careful analysis of them. The disease evidently is not so rare as these statistics would indicate. I have had a number of oral reports, and it seems unusual to find a surgeon of experience who has not observed a case.

When a hernia is present in cases of generalized tuberculous peritonitis the sac is generally involved. The affection of the hernial sac presents merely a continuation of the main process. In

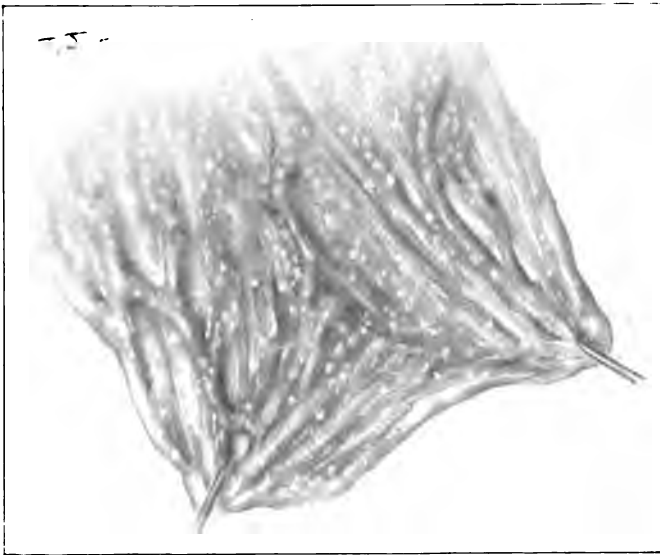


Fig. 209.—Tuberculosis of a hernial sac. No other area of disease could be discovered save in the lungs.

fact there is no record of a case in which the sac was not involved.

The degree of involvement of the hernial sac is variable. In some cases it is specifically stated that the sac was so thickened that a tumor was produced, as in a case reported by Stauber. The reason for the greater intensity of the disease in such situations may be found in the irritation produced by the contents of the hernial sac. If bacteria are free on the surface of the peritoneum they tend to seek the lowest level.

In most instances recorded the hernia involved was inguinal.

Andrews reports a case in which the process was localized in a femoral hernia. I once saw an umbilical sac that appeared to be tuberculous, but examination in the laboratory proved the case to be a pseudotuberculosis.

To the list of genuine cases I can add one observation (Fig. 209). This patient had long been the victim of pulmonary tuberculosis and a right inguinal hernia. More recently the hernia became painful and he desired to be rid of it. During the operation under local anesthesia the sac was found studded with miliary tubercles. There was but little thickening of the sac. The peritoneum inside the abdomen as far as it could be palpated through the mouth of the sac was free from tubercles and there was no free fluid or other evidence of abdominal disease. The wound healed promptly and all distress was relieved. No evidence of a general peritoneal tuberculosis developed later.

Why the affection is so localized is a matter of speculation. Weigert was of the opinion that bacilli collected in this region by gravity and the irritation produced by the hernial contents produced a favorable nidus for their development. This explanation presupposes the presence of tubercle bacilli free in the peritoneal cavity which are unable to secure a favorable field for development except where the peritoneum was irritated by the hernia. There is no knowledge that justifies this assumption. Jonnesco and Lejars expressed the opinion that all cases reported began in the hernial sac and spread to the surrounding peritoneum. They do not explain why it should localize in the hernial sac in the first place. In the case observed by me the idea of Weigert was substantiated in so far as the lesions were superficial, but there is nothing in this that would preclude a hematogenous origin, for lesions produced by injecting bacilli into the blood stream have this same superficial location.

The recognition of this condition is easy after the sac is exposed. But one case was diagnosed before operation, that reported by von Braekel. Whether or not the general peritoneal cavity is involved can be determined with a fair degree of certainty by palpating the parietal peritoneum in the region of the hernial opening. If there is an absence of tubercles about the opening the affection is probably local.



Sometimes in irreducible hernias small nodules may be found covering the sac, which in a way resemble tubercles. They may be very small, nearly translucent, and give a grating feel to the finger. On the other hand, they may be dense and scar-like. On section they show a round-celled infiltration with a more or less advanced stage of cicatrization. These nodules seem to be brought about by floccular precipitates forming in the fluid exuded from the irritated peritoneum. The nodules observed result from the attempt at encapsulation of these floccular masses. The resemblance to tuberculosis may be heightened by the formation of giant cells about these flocculi.

**Diagnosis.**—The clinical recognition of peritoneal tuberculosis is difficult. Typical cases, it is true, may be diagnosticated with considerable certainty, but a great number of cases are mistaken for more common diseases, and mild cases are overlooked entirely. Löhlein states that by carefully considering the history and physical findings, the diagnosis can be made with considerable probability but never with certainty. My experience leads me to believe that some cases can be diagnosed with great probability and some may be suspected, but many will suggest themselves for the first time after the abdomen is opened. The difficulty of diagnosis is well represented by the series of cases reported by Bonet. In 30 cases a correct diagnosis was made in 8, no diagnosis in 2, ovarian cyst was suspected in 5, salpingitis in 2, peritoneal hematocele in 1, uterine myoma in 1, and appendicitis in 1. Most surgeons with an equal experience can duplicate this series of errors, I dare say, perhaps some would even be disposed to regard enviously the eight correct diagnoses.

**Bacterial Examination.**—To secure some of the peritoneal exudate and demonstrate tubercle bacilli in it is of course the ideal method of procedure. The fact that an exudate may be unsuspected, unprocurable, or absent, lessens the value of this test. Even in those cases in which exudate is available for examination, the bacilli can not always be demonstrated. As a matter of fact in known cases of peritoneal tuberculosis, the bacilli can be demonstrated in not more than 50 per cent of the cases. The most certain method of demonstrating the bacilli is by injecting the fluid into the peritoneal cavities of guinea pigs or rabbits (Maurange). Even this

test may be negative, probably because bacilli are not present in the free fluid. Negative findings are most frequently in the purely exudative, chronic type. Rubbing up a bit of excised tuberculous tissue and implanting it directly into the tissues of a guinea pig may secure a positive result when other methods fail. Thus Courmont secured generalized tuberculosis in a guinea pig by implanting caseous material of the tuberculous serosa when the injection of the serous exudate was negative.

*Tuberculin Reactions.*—The reaction from the inoculation with tuberculin is usually untrustworthy, because in the majority of cases tuberculosis exists, or has recently existed, in some other region of the body. In instances where there is no other tuberculous lesion the evidence secured by these tests may be regarded as suggestive or confirmatory. Faludi recommends it, and reports a case in which tuberculosis was excluded by this test, autopsy showing it to be a metastatic sarcoma. Nothnagel regards it as safe and helpful. On the other hand, Henoeh is skeptical of its value and Herzfeld condemns it as dangerous, having seen miliary tuberculosis, which he ascribed to the test, follow its use.

*Chemical Analysis.*—The fact that the peritoneal exudate in this disease is the product of a reactive process, makes it possible to distinguish this disease in some instances from ascites due to static conditions. Tuberculous exudates may be higher in specific gravity than the average, or a reactive process due to tuberculosis may be complicated by a static exudate, when the changes in the peritoneum are sufficient to produce an obstruction to some of the mesenteric veins, and thus reduce the specific gravity. On the whole differentiation by the specific gravity is as apt to confuse as to clarify the problem.

The albumin content is greater in the tuberculous process than in dropsies. Helmrich gives the average as 3 to 5 per cent. The albumin content is subject to the same variations as the specific gravity. In one of my patients it was 3 per cent.

A number of cases are reported in which the exudate is referred to as purulent. Reyburn mentions a case in which a gallon of purulent material was removed. Robinson records a case in which a "large amount" of purulent fluid was removed. Köppen reports a case, probably tuberculous, in which a purulent exudate was

noted. But in none of these cases was the diagnosis satisfactory.

The distinction between a peritoneal tuberculous exudate and the contents of a parovarian cyst can likewise be differentiated with but little certainty by chemical means.

The cell content of the peritoneal exudate in tuberculosis is apt to be richer in small mononuclear leucocytes than that of the static or carcinomatous exudate. The finding of large mononuclear cells may indicate derivation from either tubercle or carcinoma nodules.

Judd suggests a method of differentiation applicable at the operating table worthy of trial. He pours peroxide of hydrogen into the peritoneal cavity, following it with saline solution. The peroxide produces a frosted appearance of the surface, which when removed by the saline, leaves the unaffected portion a normal pink color. The tubercles stand out as pearly white on a pink background.

Tuberculous peritonitis must be distinguished from diseases attended by the cardinal symptoms of this disease, notably, rise of temperature, peritoneal exudation, and tumor formation.

The leucocyte count should aid in differentiating tuberculosis from acute suppurative diseases. How unreliable it is, a number of instances in the literature will illustrate. Coves reports a case diagnosed as an "acute abdomen," having in mind a typical perforation, an appendix with diffuse peritonitis, or perforated gastric or duodenal ulcer. There were 15,000 leucocytes. At operation the intestinal coils were everywhere adherent, and the peritoneum was studded with tubercles. Schley reports a case simulating acute appendicitis. Körte and Herzfeld operated on a case of ascites following measles, and found the peritoneum studded with tubercles. The patient's cervical lymph glands were breaking down.

Fever may be present in tuberculous peritonitis. It is usually highest in the evening and often normal or subnormal in the morning. The fever is often slight as compared with the other evidence of disease. It may run a course quite like typhoid, particularly if the abdominal disease is but a part of a generalized tuberculosis. The absence of the Widal reaction and rose spots aid in differentiating it from typhoid fever. Diarrhea usually attends the acute type of tuberculous peritonitis, but it lacks the pea-green color of ty-

phoid fever. This, together with the abdominal distention and pain, invites confusion in tuberculous peritonitis. The history and the presence of other tuberculous foci may furnish a clue. In a case of my own in which this train of symptoms developed soon after recovery from measles, in a delicate child, the presence of tuberculous lymph glands in the neck gave the clue to the proper diagnosis. Symptoms of a like character following a conservative operation on a tuberculous epididymitis likewise once awoke me to the impending disaster. Pneumonia and la grippe may produce symptoms referable to the abdomen in children which may simulate tuberculous peritonitis. A slumbering retrocecal appendicitis may do the same; in fact, an appendicitis may be implanted upon an ileocecal tuberculosis, as I once discovered postmortem.

A large collection of fluid in the abdomen suggests tuberculous peritonitis in children and hepatic cirrhosis in the adult. Tuberculosis may complicate cirrhosis. Jaundice is more common in cirrhosis than in tuberculosis, but jaundice may be caused by pressure of enlarged glands on the bile ducts in the latter disease. Cases of this kind have been reported by Florand. A case of obstruction due to constriction of the common duct by adhesions has been reported by Dujon. In cirrhosis there should be an enlarged spleen.

The primary hepatic lesion may be a syphilitic affection. Other evidence of syphilis may lead to the correct diagnosis. Difficulty in diagnosis is increased in cases in which the tuberculous lesion is more extensive in the region of the liver, or the two diseases may coexist. Five of Friedländer's 88 cases were complicated by liver cirrhosis.

The subcutaneous veins are distended in 4.4 per cent (3 out of 69 cases), according to Rotch and are due to obstruction of the inferior vena cava. The veins are situated about the umbilicus. The distended veins in adhesive pericarditis are over the lower part or the whole of the chest and are not particularly pronounced about the umbilicus. There may be a history of or other evidence of a cardiac affection. Rolleston states that the ascites is more persistent in pericarditis and there is no fever. Gee, Fisher and Lazarus-Barlow report cases of ascites in primary obliteration of

hepatic veins. In such cases the site of the dilated vessels is the same as in tuberculosis.

Tumors, particularly multiple carcinosis, may be confused with tuberculous peritonitis. Exploratory incision may be required to decide the question. Even then, the differentiation may not be easy. Usually in carcinoma there is greater tendency toward confluence of lesions and the lesions are often umbilicated. History suggestive of a primary malignant disease, particularly of the stomach, is helpful. Peritoneal metastases from ovarian tumors, particularly papillary cystadenomata, are apt to simulate tuberculous peritonitis. Tumors of the abdomen may resemble sacculated tuberculous peritonitis, or the thickened omentum may simulate a solid tumor. The omental tumors are prone to lie in the region of the colon, even between the colon and the stomach. These are sometimes attended by gastric disturbances, such as vomiting and pain. When associated with rapid loss of flesh, the presumptive evidence seems to lean toward carcinoma. Twice in my experience an abdominal incision was required to settle the diagnosis. In one instance a microscopic section was required to finally settle the matter. The presence of an irregular fever would suggest the possibility of tuberculosis, as would a white cell count below 10,000, while one above that figure would speak for malignancy. Morris reports two cases in which tuberculosis was mistaken for multiple carcinosis.

In one case in my experience a cystic tumor the size of a fetal head developed above the umbilicus, following a violent traumatism in this region. It was attended by a loss of 40 pounds in weight. A pancreatic cyst was diagnosticated, but operation proved it to be a localized tuberculous process. The patient had always been well previous to his injury. There was no evidence of tuberculosis elsewhere.

Hydatid cysts and cysts of the mesentery and omentum have been reported in the literature as having been mistaken for sacculated peritoneal tuberculosis.

Tumors going out from the pelvis may simulate tuberculous peritonitis. Papillary cystadenomata, as already mentioned, may rupture early and become disseminated over the peritoneum, producing an exudate with moderate pain. Pelvic examination shows a

diffuse infiltration of the culdesac, which can not be distinguished from the infiltration of a primary peritoneal tuberculosis. Paterson has recently reported a case in which a suppurating ovarian cyst resembled a peritoneal tuberculosis. If the patient is young, particularly if there is or has been a tuberculous process elsewhere, tumor of the pelvis suggests tuberculosis. A cyst of the urachus may be simulated by tuberculosis. Doran believes the allantoic cysts reported by Lawson, Tait and B. Robinson are examples of the residuum of earlier pelvic tuberculosis. In patients of more advanced years, particularly if the cyst can be well outlined, the diagnosis inclines toward malignancy. In a woman aged sixty-two, presenting the usual symptoms of a malignant tumor of the ovary, laparotomy disclosed a tuberculosis of the great omentum.

Parovarian cysts may simulate a peritoneal exudate. The pelvic wall will be found free from induration, however, in these cases.

A localized peritoneal abscess in the pelvis or a dilated tube may lead to the diagnosis of simple hydrosalpinx or gonorrheal salpingitis. I once diagnosticated myoma, which proved to be caseated tuberculous tubes.

After all factors have been considered, it is well to enter these cases on the operating room bulletin board "Exploratory laparotomy; possible tuberculous peritonitis."

**Prognosis.**—The prognosis of peritoneal tuberculosis has been discussed by a vast number of writers. The augmentation of the literature is due to the fact that the later reporters have had better results to offer. That more persons recover now than formerly is probable, broadly speaking, yet one needs to look further than the superficial statements of the literature before a fair judgment can be reached. Whether more recover or whether merely more recoveries are recognized is the question that must be kept in mind.

The older writers held the most melancholy views regarding the probable outcome of a case of tuberculous peritonitis. Aran typifies the view of the period graphically as follows: "*la terminaison constante des affections tuberculeuses de peritoine est le mort.*"

The older authors emphasize with greater consistency than more recent writers the importance of coexisting lesions in the question of prognosis. Thus Guéneau de Mussy stated that if not complicated by lesions incompatible with life the patient may recover. Louis noted

that the peritoneal affection may improve and the patient may then succumb to the lung involvement. Likewise Siredey and Danlos believed cure was possible, at least for a time. Fernet believed that the patient might recover from the subacute form and Hanot believed that cure was the rule. On the whole, however, earlier writers entertained the melancholy views of Aran above quoted.

A glance at the text books of a generation ago shows that the prognosis was regarded with as much pessimism as by the early writers above quoted. Thus Flint stated that all patients die, and v. Bauer declared that the vast majority run a fatal course. Eichhorst may be quoted as follows: "The course is nearly always unfavorable from the increasing peritoneal exudates. Against the disease itself we are altogether helpless." These pessimistic statements could be multiplied a hundredfold.

Even after the possibility of recovery was proved by König most internists were slow to admit the possibility of spontaneous recovery. Kussmaul records the recovery of an extreme case, and notes the recovery of others. Henoch denies spontaneous recovery, and believes that when such takes place simple chronic peritonitis, not tuberculous peritonitis, is present. Liebermeister and Vierordt hold the same view. These melancholy statements filled the literature up to the time König's epoch-making paper appeared. Since then the vast literature which has appeared breathes optimism. At first thought it would seem that such a radical change in viewpoint must have come because of the curative effect of surgical treatment. A careful perusal of the literature casts much doubt on the validity of such a conclusion. Operative treatment has made possible a much closer study of the disease, particularly in its earlier stages. Diagnosis is never certain without a direct inspection of the field. When it became generally understood that operative treatment was the correct procedure, the doubtful cases were subjected to laparotomy. The opportunity offered to study the disease by operative autopsy acquainted the profession with the signs and symptoms, so that diagnosis without operation became much more certain than it was in the preoperative days. The result was that milder cases were recognized by the internist. He observed recoveries and his results too became better than in the days before König's publications. So marked is this fact that many

writers now regard tuberculous peritonitis as a medical disease. At any rate whether more patients recover now than formerly or not, certainly more recoveries are recognized. No doubt the better management of the primary lung involvement saves many patients the peritoneal complications. And no doubt, too, better understanding of the conditions and generally improved environment have affected the outlook, so that a considerable prospect of cure may be offered the patients afflicted with peritoneal tuberculosis.

After the appearance of König's paper, statistics had to do with the results of surgical treatment. Some of the more carefully compiled statistics may be quoted. König secured a recovery of 65 per cent in 131 cases. He regards persons as cured who have remained free from the disease for two years. Roersch, in the collected statistics of 359 laparotomies, found recovery in 70 per cent. Margarucci in 250 cases, reported recovery in 85 per cent. Thomas in 346 laparotomies reported 73 per cent cures in the exudative type, 57 per cent in the dry, and 57 per cent in the encapsulated type. V. Krencki in 266 laparotomies reported 71.65 per cent cures in the encapsulated type. Baumgart in 54 cases had 17 per cent recoveries and 8.5 per cent improvements. Frank in 63 personal cases had 55 per cent recoveries in the exudative, and 21 per cent in the dry type. In the ulcerative, suppurative form Frank lost all of his three cases, while Thomas had a recovery of 70 per cent of his cases of this type. Schramm in 45 cases, treated 25 expectantly with 36 per cent deaths, and operated upon 20 with 10 per cent deaths.

König places deaths from the operation itself at 3 per cent, Schmitz at 10 per cent, and Meyer at 10.8 per cent. Meyer got better results by laparotomy in males than in females. Shattuck reported on 98 cases observed for periods of from 2 to 11 years. Of these, 57 could be traced. Of the 57, 68 per cent of the medically treated patients were dead at the time of the compilation of the report, and but 47.3 per cent of those surgically treated.

Pic secured data by observing a considerable number of cases untreated. Thus in 64 cases of young girls, 50 per cent died, while but 4 per cent recovered. Those readers who have a liking for statistics will find them collected by Faludi, together with the literary citations.



Many of the papers presenting glowing prognostic possibilities bear the earmarks of subjective enthusiasm.

Rose is obviously right when he contends that statistics have been prejudiced in favor of operative treatment. The reported cases are small series, and in many instances selected cases only have been subjected to operation. On the other hand, those who report spontaneous recoveries have included cases which were not tuberculous in nature at all. To obviate this difficulty, therefore, it is necessary that those contending for spontaneous recovery prove their diagnosis by demonstration of the bacteria. This has been done for small series, notably by Borchgrevink.

In some of the series reports have been made too soon after operation. Healing of the incision does not constitute a cure. As long as two years after apparent cures relaparotomy has still showed the presence of tubercles. In König's statistics of 65 per cent cures, only 24 per cent had remained cured for two years or longer. In Margarucci's compilation of 253 cases reported as cures, only 26 per cent had been operated on more than a year before. Roersch reported on 358 cases with 70 per cent cures. Only 15 per cent, however, had been operated on 2 years or more before. In Adosides' 405 cases, 15 per cent had been cured more than 2 years.

The cures from expectant treatment furnish a no less imposing series of statistics than those from operative treatment. Frank reports 63 operative and 8 nonoperative cases in Czerny's clinic. Those surgically treated showed 38 per cent recoveries; those expectantly treated, 50 per cent recoveries.

Rose reports on 71 cases medically treated, all in patients over 14 years of age. Of these 9 died while in the hospital. In 52 cases which he traced, 34 died after leaving the hospital, one remained sick, and 16 had recovered. In his series there were recoveries in 36 per cent in the tumorous form, 33 per cent in the ascitic, and 29 per cent in the mixed. He noted 50 per cent in the acute form, 31 per cent in the subacute, and 16 per cent in the "schleichende."

Frank had the best results in the exudative form, namely, 40 to 50 per cent recoveries. In the adhesive form 25 per cent recovered. The prognosis in the ulcerative and suppurative forms is very grave. The best prognosis is seen in women in whom the disease began in the adnexa, and who were treated by removal of the

adnexa,—75 per cent recoveries in the exudative, and 50 per cent in the adhesive. The fecal fistulas present a very bad prognosis. Other statistics may be recorded as follows: Borchgrevink had 22 cases of which 19 recovered; Monti 10 cases of which 6 recovered; Schmitz 32 cases of which 30 recovered; and Sutherland 27 cases of which 22 recovered.

Bonet explains the diversity of results reported by the various writers as due in part to the difference in the classes of patients treated. In the poorly nourished and those living under unfavorable hygienic conditions, recovery naturally is less likely than in those whose constitutional and environmental conditions are more favorable.

Wright makes the point which every observer must have noted, that even when patients apparently recover from their peritoneal affection, they are prone to die from tuberculosis of some other organ.

The statistics above quoted will convey as much information as a more extended series. The value of statistics has often been questioned and nowhere is the occasion for this more warranted than in the statistics relative to the results of any treatment, particularly a treatment that has not had the criticism of the profession for at least 10 years.

Considering the late statistics only, it seems a fair estimate that some 30 to 50 per cent of cases of tuberculous peritonitis recover either after operative or expectant treatment. Cure could not be spoken of in any instance, because the direct relation of the therapeutic measures employed and the subsequent recovery of the patient has not been determined in any case.

**Conservative Treatment.**—As in tuberculosis of other regions of the body the chief agent to be directed against the disease are the natural defensive forces of the body. These may be classed under conservative treatment. Not infrequently definite conditions arise where active operative interference is warranted. When abscesses form active intervention is imperative. Whenever it is possible to remove the focus of infection operation offers a prospect of relieving the patient of a part of his burden. These various phases may be considered in detail.

*Medical.*—The exhibition of drugs for the cure of tuberculous

peritonitis is a trail which runs quite parallel with the medicinal treatment of this disease located in other organs. The period in which any paper or book was written can be quite accurately determined by observing the measures recommended. For instance, formerly codliver oil and the hypophosphites received the most frequent mention, pressed closely by creosote and its congeners.

A few variations are recorded. Sandler gave mercury with chalk and iodoform, and applied the same in the form of an ointment over the whole abdomen. Schmidt used cacodylate of soda. Mühlberg applied stupes of cinnamon oil, ten drops to a dram of olive oil. Millard applied collodion to the entire surface of the abdomen. Green soap was in quite general use, particularly in Germany, before laparotomy became the common mode of treatment. Soap may be a good prophylactic, but it is difficult to understand how it could be curative. Wilcox used inunctions of iodoform in ether and oil with success in two cases. Hofmann used iodine locally in four cases, with success in all.

*Climate.*—Lalesque reports a cure in five cases by a prolonged residence at the seashore; and Leroux is of the opinion that sea air is particularly useful after the acute symptoms have subsided.

*X-ray.*—Porter was the first to give this method a systematic trial. Ausset and Bédart had previously reported the cure of a case by this means. Bircher reported on 16 cases treated by x-rays after operation. Of these 7 were cured and 5 were improved. Of the 7 reported as cured, only 4 had been well for a year or more. Twelve cases were treated by the x-rays alone. Of these 6 were cured, and 2 were improved. This investigator used hard or medium-hard tubes, treating the patient daily for three or four weeks, the treatment lasting from fifteen to thirty minutes at each sitting. If no results were produced, an interval of two weeks was allowed to elapse, and then the treatment was repeated. Shober advises the use of the x-rays in conjunction with laparotomy. The researches of Falk made upon animals indicate that wound healing is not interfered with by radiation immediately after operation. This combination of incision with radiation under modern methods would seem to be worthy of a trial. It must be remembered, however, that theoretically x-rays are contraindicated, if the theory of healing by fibrosis is correct. The x-rays cause a degeneration

of the connective tissue, and this might be expected to retard the healing process.

The reports available fail to show any marked influence of the x-rays on the course of the disease.

Ausset and Bédart report a case of cure after using x-rays for eighteen months, after conservative treatment and laparotomy had been used.

Sun baths, as suggested by Oppenheimer as a corollary to the use of the x-rays, may be mentioned. He reports favorable results in two cases. He believes that the action of the sun's rays produce a hyperemia of the peritoneum, acting in this way like a laparotomy.

*Tuberculin.*—Varnek reports a cure from the use of tuberculin. Anderson also records a case. Bumm reports on its use in conjunction with laparotomy. McDonnell reports the use of Marmorek's serum, 5 c.c. each week for six weeks.

*Paracentesis.*—Von Mosetig-Moorhof recommends simple puncture. Mader recommends puncture with a careful pressing out of the fluid, and the subsequent use of abdominal compresses with the idea of producing a slight inflammation. Seganti punctured the abdomen at two points, and irrigated the cavity through these openings with salt solution. Mathis regards puncture with lavage as the best treatment. Schömann recommends drainage with subsequent iodoform injection. He begins with 1 to 2 c.c. of a 1 per cent emulsion, and increases the dosage with each renewed injection. The injections are repeated every four to eight days. He treated seven cases, which he regarded as cured after three to ten weeks. Rendu injected naphthol camphor intraperitoneally after draining off the fluid. He used five Pravaz syringefuls. This treatment is evidently a bit heroic; at any rate, it was followed by nausea, pain, and vomiting. Von Helmrich reserves puncture for cases in which the distention is so great that digestion is impaired, or edema of the lungs is threatened. Gusserow sums up the dangers of puncture as follows: sepsis, following puncture of a gut and possible injury to blood vessels. He notes that complete emptying of the fluid is not possible by this means.

*Air.*—Following the belief that it was the air that produced the beneficial effect in laparotomies, numerous operators attempted to

secure these benefits by blowing air into the peritoneal cavity. Among these may be mentioned Folet, Durán, and von Mosetig-Moorhof. Brial collected eleven cases treated by this method. Nolen constructed a special apparatus for applying this kind of treatment. Napoleone reports the recovery of a very grave case after the injection of air into the peritoneal cavity. Floris reports three successful cases; and he advises the introduction of a volume of air equal to that of the fluid removed. McGlinn inflates the abdomen with oxygen, allows it to escape, and inflates it again. This process is repeated a number of times.

**Operative Treatment.**—*Danger of Operation.*—Rolleston states that an operation is not indicated in any case before a year from the onset, and that it is unnecessary in the ascitic and fibrous forms in the absence of symptoms of intestinal obstruction, but that it is indicated in abscesses and intestinal obstruction. Thoenes emphasizes the danger of the operation *per se* because of collapse, sepsis, fecal fistulæ, etc.

Pic regards fever, lung tuberculosis of even moderate severity, and intestinal ulcers as contraindications to operation; and Elmassian agrees with this opinion.

*Time for Operating.*—Since König first reported cures in four cases treated by laparotomy, this has been the favored method of treatment. The question of the time at which laparotomy is most effective, has engendered much dispute. Gelpke, on the basis of animal experimentation on dogs, concludes that early laparotomy is not followed by the best results. He regards the third or fourth month as the best time for operation. He believes that the exudate is at first beneficial, in that it contains a tubercle antitoxin which is alone sufficient to produce a cure in mild cases. Bonet believes that operation is contraindicated in acute cases. Friedländer agrees with this opinion, and recommends operation only after the acute symptoms have subsided. He believes that early operation is useless, and is seconded by Gatti, who holds that at this stage the bacilli are still too virulent to permit such a procedure, but that later they undergo involution.

*Technic.*—Frank advises an incision twelve or fifteen centimeters long, reaching from the umbilicus to the pubis. Baumgart, on the contrary, advises a short incision, and in females a vaginal

section. The advantages of the latter are that infection is less apt to take place, there is less danger of scar hernia, and the tubes can be removed by this means, and the pathologic tissues reached readily, the involvement being usually the most pronounced in the fossa of Douglas. Löhlein had previously advised posterior colporrhaphy for better palpation of the pelvic peritoneum and for the excision of a bit of peritoneum for the purposes of diagnosis, as well as to establish drainage. Frank opposes this route, because it is useless in the adhesive form, may endanger the bowel, and does not permit an inspection of the entire peritoneal cavity.

Frank, in common with many others, advises tamponage and drainage in cases in which there is much bleeding or pus, in which cavities not obliterated by fluids exist after the escape of fluid, and in the serous suppurative form.

Most operators regard drainage as dangerous because of the likelihood of forming a fecal fistula. Many of the earlier operators used a drain, for a longer or shorter period. Miller used a rubber tube and an iodoform-gauze drain, allowing them to remain in for two months. Briddon employed drainage and tamponage. A modified form of drainage was employed by Evler. He stitched the peritoneum over the edge of the recti muscles in an incision below the umbilicus, and then closed the skin, hoping in this way to secure drainage into the subcutaneous tissue.

Martens and Lindner regard drainage as superfluous. Gelpke notes that fecal fistulas may arise after drainage.

Thoenes recommends an energetic rubbing of the peritoneal surface during laparotomy in order to hasten the natural tendency to encapsulation. Judd used peroxide of hydrogen. Frank advises a thorough sponging out of the peritoneal cavity during laparotomy. Hofmann treated four cases by painting the visceral and parietal surfaces with iodine. All recovered. In none of these cases was the disease severe, and too short a time had elapsed after operation to permit an opinion. Experiments on animals have proved that such a remedy must be used with great caution, for a small amount of iodine in an animal's abdomen may produce death.

Riva advises irrigation with eight to ten liters of salt solution. Wilcox tried rubbing in iodoform. Jordan tried irrigating with thymol, boric acid, etc. Von Marchthurn secured a recovery of 21

out of 38 cases, or 55 per cent, by this means. Eccles advises the use of dilute iodoform in oil. The patient recovered both from the iodoform and from the disease.

Repeated operations have been recorded by many operators. Galvini reports a case in which laparotomy was done five times, with recovery.

*Management of Adhesions.*—Both Friedländer and Thoenes advise against the separation of adhesions because of the danger of producing a fecal fistula. Porter advises the separation of slight adhesions if necessary to let in light and air.

*Removal of Tubes.*—Kaulich was the first to emphasize the importance of the removal of the primary focus. Veit recommends the removal of the tubes in all cases, whether primarily or secondarily involved. Runnels advises that the primary focus be searched for diligently and if found, removed. Winter and von Kreneki found that 66.1 per cent recovered without the removal of the tubes, and 76.6 per cent with their removal. Mayo secured 25 recoveries in which the tubes were removed. In 7 of these simple laparotomy had already been performed. Robson advises the removal of not only the tubes, but also of the affected glands and even of portions of the gut. Stone in 122 cases did not find the tubes involved in any. In girls the tubes are seldom involved. Maas, quoted by Murphy, could collect but 8 cases. Goodall believes that in 99 per cent of the cases in which the tubes are involved removal is necessary, though in 30 to 50 per cent he could not locate the primary focus. More recently a more conservative note has been sounded. Stone believes that infected tubes do not require removal any more than in infected omentum or intestine. Tweedy reports a case in which conservative treatment was followed by pregnancy. Heimann advises against the removal of the tubes, and in his case reports records good functional results by conservative treatment. My own experience is in entire accord with this more conservative view. I had one patient who conceived after loosening adherent tubes. Where the tubes are deeply imbedded their removal may hazard a fecal fistula. In the exudative type, in which the tubes are easily accessible, their removal can not hasten the cure. In the primary and isolated lesions of the tube, where local thickening with caseation is present and where

the remainder of the peritoneum is free, removal is in order. Finally, tubes are removed for tuberculosis when the disease does not exist.

*Removal of Tuberculomata.*—Tuberculomata have been removed in three instances. Beatson removed a mesenteric tumor from a child aged four; and Kukula removed one in a man of thirty-eight. Baum removed a mass from the omentum the size of the palm of the hand over the pyloric region with the attached gut. A pocket of glands and many small tumors remained in the region of the main tumor.

*Operation in the Dry Form.*—Most authors advise against operation in the dry form. Von Helmrich sees in the tumorous form a positive contraindication, because no good can come from it. Gelpke also advises against it because of the danger of fecal fistula. Göfert advises it only in cases of encapsulation and intestinal occlusion. Schramm advises against operation in general in the dry forms, but admits its use in such cases as begin in the tubes, since in these instances the primary focus may be removed.

*Objections Against Operation.*—As in most methods of treatment, the benefits claimed were, no doubt, much exaggerated. Soon the results began to be questioned, and vehement opposers were not lacking. Among these may be mentioned Comby and Grange.

Friedländer reports on 20 cases who died following operation, and two had fistulas. Herzfeld in 11 fatal cases had 5 fistulas. Of Borchgrevink's 12 cases 3 had fistulas. These statistics are sufficient to show that operation promotes the formation of fistulas.

Regarding the question of the possibility of a secondary infection of the abdominal wall after laparotomy by the tuberculous process, Parker Symms answers in the negative. He believes that tuberculous infection of the wound does not take place. Braun, however, records a case in which such an infection reached the surface by way of a stitch tract. Lindner is probably correct in stating that such an accident is very unlikely to happen, as the through-and-through method of suture has been abandoned. In one of my cases, a woman of twenty-three, with a massive tuberculosis of the pelvic organs, infection of the wound tract took place in which healing was not secured for more than a year. Healing of the sinus, as



well as cure of the original disease, finally occurred, however, and was complete five years later.

Lindner's collected statistics contain reports of operations in 205 cases, in which there was an operative mortality of 7.5 per cent.

*How Operation Does Good.*—About twenty theories have been advanced to explain in what way operation does good; and this in itself is enough to throw doubt on the value of the procedure. Jaffé and Friedländer have advanced the theory that it is the adhesions produced by operation that promotes recovery. Jaffé made the important observation in five cases in which he had occasion to reopen the abdomen that, while there was a clinical cure, the tubercles remained. Jordan reports a similar case. Friedländer makes the point that in the case in which improvement takes place only after a long interval following operation the recovery can hardly be ascribed to the operation. These cases, though small in number, are sufficient to show that cure can not be ascribed to the operation in all cases recovering after operation, and that some cases that appear clinically to have recovered have not recovered anatomically.

The most generally accepted explanation of the value of operation is that an active and passive hyperemia is produced. Gatti, Nassauer and D'Urso have particularly emphasized the validity of this explanation.

Fritsch believed that laparotomy has the effect of restoring the absorbing ability of the peritoneum by restoring the circulation. As evidence he cited the increased diuresis following operation. This restored circulation, according to Nassauer, increases the nutrition of the tissues.

Hildebrandt found from animal experimentation that the arterial hyperemia disappears after a short time, but gives way to a venous hyperemia lasting several days. Lohmann, besides a hyperemia following operation, hypothesized an increased diapedesis of leucocytes. Pitfield believes that it is the blood that gets into the peritoneal cavity that exerts the beneficial influence after operation. Sippel believes that air contact is the important factor, for when the operation was done under a normal salt solution cure did not result.

Schegoléff tried to arrive at a conclusion by means of animal ex-

perimentation. He produced tuberculous peritonitis in dogs, and then studied the effect of laparotomy. He concluded that in the early stages the disease is cured by this means, but in the later stages it is not. He ascribes the cure chiefly to the reaction produced by the operation. More leucocytes and phagocytes, together with an active proliferation of the connective tissue, result. Physical agents, too, have some influence, according to this author. Among these he mentions heat, air, and perhaps light. These act by increasing the irritation. Kishenski, from dog experiments, concludes that laparotomy increases the proliferation of connective tissue about the nodules. As a result of this ring the center caseates, and later becomes calcareous.

Finally, it must be remembered that laparotomy may secure improvement in conditions when cure is out of the question, as in cancerous peritonitis following ovarian carcinomata, as noted by Freund.

### **Pseudotuberculosis**

Under this caption may be included a heterogeneous group of diseases which produce lesions or clinical manifestations resembling those characteristic of tuberculosis.

These may be classified into (1) those due to organisms other than tubercle bacilli, (2) those due to foreign bodies, and (3) those characterized by a chronic reactive condition, nontuberculous in nature, due to various or indeterminable causes.

1. *Bacterial Pseudotuberculosis*.—In this group are those caused by the bacillus pseudotuberculosis rodentium (compare Ophüls), and those caused by a related bacillus resembling the above, of which two cases have been reported by Du Cazal and one by Wrede. A diphtheroid bacillus has been the cause of lesions resembling tubercles. Flexner reports an interesting condition under the name "pseudotuberculosis hominis streptothrica." The patient was a male, aged 70, who had died with symptoms of pulmonary tuberculosis. The autopsy showed the omentum rolled up beneath the transverse colon. Translucent nodules of various sizes resembling tubercles were irregularly scattered over the surfaces of the peritoneum. The liver and spleen showed similar nodules. A branching organism was discovered on microscopic examination. Mould

fungi have been accused of playing a similar part. Finally, the organism specific of blastomycotic dermatitis may be classed in this category.

2. *Foreign Body Tuberculosis*.—First in this group may be mentioned those lesions developing about animal parasites and their eggs. Helbing records a case in which, during an operation for the removal of the appendix, tubercles were found on the cecum. Section of these revealed eggs of the *tænia*, which had obviously escaped after perforation of the appendix. Dévé reports four cases in which pseudotubercles developed about hydatid hooklets or pieces of hydatid membranes. A number of interesting cases have been recorded in which pseudotubercles formed about a variety of foreign bodies. Meyer records a case in which pseudotubercles developed about cholesterin crystals which reached the peritoneal cavity by the rupture of an ovarian cyst. Von Recklinghausen (quoted by Meyer) reports a case in which bits of sponge furnished the basis for a pseudotuberculosis. Hanau and Cooper report cases in which pseudotubercles were found about an old gastric ulcer, the basis of which was formed by food particles. I have seen a similar case, in which bismuth, exhibited for the cure of the gastric ulcer, furnished the basis for a small crop of tubercles about the adhesions in a perforating ulcer.

3. *Chronic Idiopathic Nontuberculous Peritonitis*.—Those cases which resemble tuberculous peritonitis clinically may be placed in this group. For the most part there is little anatomic resemblance to tuberculosis, but in some instances there may be a general thickening of the peritoneum and subperitoneal tissue without the production of tubercles.

Before laparotomy became the prevailing method of treatment for peritoneal tuberculosis, many cases of chronic peritonitis were assigned to the so-called idiopathic group. At one period this variety was thought to exceed the tuberculous in frequency. The increased opportunity which laparotomy gave for observing chronic peritonitis, on the other hand, reduced very much the proportionate number of the idiopathic variety. To such an extent is this true that some recent observers deny the existence of an idiopathic variety entirely. There is no doubt that the idiopathic form is very rare, but some cases have been observed which can be placed

under the tuberculous category only on negative evidence. The safest way, therefore, is to recognize our limitations, and retain this group for cases of chronic peritonitis in which it is impossible to diagnosticate tuberculosis on positive evidence.

On the other hand, the older writers were unquestionably wrong when they placed in the category of idiopathic peritonitis all cases running a course identical with tuberculous peritonitis, but terminating in recovery. It has of course been abundantly proved that cases of tuberculous peritonitis may recover.

An analogue for idiopathic peritonitis has been sought in idiopathic pleurisy. Many observers hold that all exudative pleurisies are tuberculous in nature. This has not been proved. Such broad statements may be of great convenience, but they form a poor basis for scientific investigation.

Bauer divides chronic nontuberculous peritonitis into three groups: (1) those which are terminal to an acute process; (2) those in which the peritoneum is placed in a reactive state as the result of chronic circulatory disturbance, as is seen in obstruction to the portal vein, nutmeg liver, or adherent pericarditis; and (3) those in which there is a gradual development of a peritonitis without a demonstrable etiology.

In cases of irritation of the peritoneum by the exudate from an inflamed organ, a peritonitis may be produced which is essentially chronic in character, tending to recovery as the causative factor recovers. This may be seen about the gall bladder in cholecystitis. It has been described as "gall-bladder peritonitis." The same process may be instituted when a chronic or subacute gastric or duodenal ulcer approaches the surface. An irritant exudate is produced, and sets up a limited chronic peritonitis, which may lead to peritoneal adhesions. Similar processes may be excited about the appendix. These processes may find expression in exudation, in the formation of adhesions, or in a chronic hyperemia of the peritoneum. I once operated on a patient who gave a typical history of appendicitis. An operation done after four months showed an appendix the most of which had disappeared. A mere string extended from the cecum to a segment of appendix 3 cm. long, representing the terminal end. Obviously, the remaining portion had disappeared by a process of necrosis. Extending from

the appendiceal region mesially and upward were abundant web- and band-like adhesions and much exudate. The peritoneum was yet in a state of reactive hyperemia. No doubt this process was instituted by bacteria, but it is equally certain that tubercle bacilli were not responsible for its origin. Ruptured serous ovarian cysts may likewise produce a chronic peritonitis at a time when the papillary processes have not yet become malignant and capable of primary metastasis.

Obstruction to the venous return ordinarily does not cause notable thickening of the peritoneum. Cases have been recorded in which thickening has occurred about the site of puncture. However, it is likely that chronic obstruction may cause a reactive hypertrophy of the peritoneum, particularly if the lymph return is likewise interfered with. The process then becomes analogous to elephantiasis in the extremities. At least this is my interpretation of a case of adhesive pericarditis in which laparotomy was done to relieve the accompanying ascites. In this case the peritoneum, particularly the parietal in the region drained by the mammary vein, was distinctly thickened, and mounted by small granular elevations. These, however, were composed of lymphatics, and the thickened peritoneum resembled in structure an ordinary elephantiasis.

Cases are recorded in which there has been extensive hyperplasia of the subperitoneal tissue without any known cause. As a clinical example of this may be mentioned the case reported by Porter.

A similar case was reported by Henoeh. This case was that of a girl, aged ten, in whom, following an injury, an exudate formed and required puncture twice. After puncture a tumor the size of the hand formed. At autopsy a simple peritonitis was revealed. In many places the serosa was enormously thickened, the peritoneum measuring in some regions 0.5 to 1 cm. It is conceivable in this case that an injury to the gut wall permitted the exit of a mild form of infection.

I have seen such areas of thickened gut, and have been impressed by the histologic resemblance of such tissue to the tissue of woody phlegmon of the neck.

Cases in which a chronic exudative or exudative-adhesive process

begins without demonstrable exciting cause, and which in the main resembles a mild tuberculous peritonitis, constitute the majority of the idiopathic cases recorded in the literature.

The recorded cases present a great variety of conditions. Some of them recorded as idiopathic were unquestionably tuberculous, while others would better be classed in the group of polyserositides. For example, Stitzer and Rochs report the following case: a girl, aged fifteen, who had had abdominal disturbance since an attack of measles seven years before, and had been punctured in her eleventh, twelfth and thirteenth years. Each time a large quantity of greenish yellow fluid was removed. Death finally occurred with progressive edema, icterus, and cyanosis. At autopsy the peritoneal cavity contained a large amount of colloidal fluid, and yellowish exudate in part cloudy. The abdominal organs were adherent into a convoluted mass, and were joined to the thickened parietal peritoneum. A nutmeg liver was adherent to the organs lying near it. Even more striking is Steinbrück's case in which after terminal phenomena similar to the above, the lower part of the abdominal cavity contained sacculated exudate, and the omentum and transverse colon formed a cavity above. The intestines were adherent to each other, and the liver was encased in a fibrous capsule. In addition, the pleura were adherent, and there was a synechia of the pericardium. A similar case is recorded by Riedel. On the other hand, Heubner reports a case the idiopathic nature of which may well be questioned. A boy after some years of abdominal complaint became ill with a pulmonary affection attended by fever which terminated fatally. Autopsy showed adhesions of the intestines without tubercles, and both old and recent tuberculosis of the lung. In the light of our present knowledge there can be but little doubt that this represented a peritoneal tuberculosis.

The vast majority of cases have to do with clinical records in which the patient recovered. Such cases must of course be involved in the greatest doubt, since we now know that many cases of tuberculosis recover. Thus five of Steinbrück's six cases recovered. Quinke noted that females near puberty were the most frequently affected, a fact which we now know to be true of tuberculosis. Vierordt remarks on the difficulty of ascribing any

causative factors. Winge likewise could suggest no etiologic factors. His fourteen cases, it is worth noting, were all clinical observations, in none of which was tuberculosis excluded. In fact, he expressly noted the frequent coexistence of pleurisy. Fränkel, while not doubting the existence of this type of peritonitis, comments on the difficulty of making a positive diagnosis in cases which recover. Hagelstam and Delpuech ascribe as etiologic factors such general conditions as uremia, malarial poisoning, lead poisoning, and alcoholism. Hagelstam believes that cases arising after acute infectious diseases are due to the action of the toxin upon the peritoneum, but not to the action of the bacteria themselves. Other reported cases in which material obtained at operation or autopsy was examined for tubercle bacilli, offer greater difficulties for judgment. Thus Spaeth and Prochownick examined tissue and declared against its tuberculous nature because of failure to demonstrate tubercle bacilli. These cases clinically were tuberculous, as the authors thought, and only the negative bacteriologic study caused them to place their cases in the category of idiopathic peritonitis. That these investigators failed to find the bacilli can not occasion surprise, since in many known tuberculous lesions this is impossible, and it is only by the aid of animal inoculation that a diagnosis can be made.

The clinical symptoms of the tuberculous and idiopathic types as given by the older authors, are essentially the same. Henoch does maintain that in the idiopathic type the exhaustion of the patient is less. Vierordt gives the specific gravity of the exudate as between 1.017 and 1.027, and the albumin content between 4 and 7.5 per cent, figures which correspond with those generally accepted for tuberculous peritonitis.

After König's treatment by laparotomy was generally adopted the report of idiopathic cases rapidly became less frequent. Reports of specific cases practically ceased, and what mention is found in the more recent accounts, speaks of it in such general terms, as "rare disease," etc. Borchgrevink proved by means of animal inoculation that the simple forms are in fact tuberculous in character. This author advises the abandonment of the designation "idiopathic peritonitis" entirely. At the same time, he admits that there are other irritants, for example, chemical, which may

produce a serous exudate; but these, he claims, have an altogether different significance, and are not comparable to tuberculous peritonitis. In this he is unquestionably right. However much one might want to expurgate the literature of all the recorded cases after reading them, it may, nevertheless, be well to retain a class for cases in which a tuberculous nature is not capable of demonstration, which may now be "idiopathic," but after more careful study may reveal their true nature.

**Polyserositis.**—A very remarkable condition of the peritoneum, known as *polyserositis*, may be referred to here, because, so far as is known, it may represent a corollary to the idiopathic peritonitides above mentioned. In this condition the peritoneum, both parietal and visceral, as well as the pleura and the pericardium, become more or less thickened, and ascites is present in greater or less degree. The onset is often brusque, affecting persons of previous good health. The thickening of the peritoneum, which may be massive, is made up of fibrous tissue without evidence of degeneration. Gangitano has noted a distinct endarteritis. In the single case I have been able to examine, there was an obliteration of many vessels, and, judging from the character of the tissue, no doubt the lymphatics likewise were disturbed. My judgment is that the lymphatics are primarily affected. The cause is not known. Huguenin thinks "it has something to do with tuberculosis," and Pancet thinks that it is due to a tuberculotoxin. Von Creigern, on the contrary, denies this for his patients were negative to both Wassermann and tuberculin tests. Trauma has been blamed by Gazette and Gangitano. Primary affections of the liver with subsequent congestion have been suggested (Hübsmann), but Esau reports a case in which the liver was not affected. This condition is one, therefore, in which the serous membranes become thickened, and in which there is an associated exudate for which no cause can be ascribed.

Kieseritzky believes the condition may be due to either a primary serous inflammation with subsequent circulatory disturbance or to a primary pericardial affection.

There are several conditions related to the hyperplastic processes of the peritoneum. Among these may be mentioned linitis plastica and sclerostenosis of the stomach. For literature see Krompecher.



### Chronic Hyperplasias of the Peritoneum

Either alone or in association with other serous surfaces the peritoneum, in rare instances, undergoes marked thickening. In yet rarer instances other serous surfaces may undergo such thickening, and the peritoneum remain unaffected. In conformity with the multiple character of the lesion it has been designated "multiple serositis." The frequent predominance of the pericardium led Pick to call it "pericarditic pseudocirrhosis of the liver." Curschmann emphasized the liver involvement designating the affection "Zuckerguss-leber," which is translated by Kelly as "iced liver." The French call it "perivisceritis." American cases seem to be very limited. Osler, Cabot, Herrick, and Kelly seem to mention about the only recorded cases. The last-named author gives a resume of the literature, and tabulates all the known cases, 39 in all.

The essential nature of the lesion is a matter of question, as the multiplicity of names employed by the various writers to designate it indicates. We need be concerned here only with the change within the abdominal cavity. Pick believed the changes in the peritoneum to be the result of long-continued congestion and the resulting persistent ascites. Weiss believed that there is an intervening hepatic disease, in which this organ and the peritoneum suffer alike, though in varying degrees. The peritoneal changes, according to one group of men, of whom Pick is the most prominent, are of the opinion that the changes in the peritoneum are secondary to the liver changes, and these, in turn, are due to long-standing congestion. Others, of whom Weiss is the most conspicuous, believe that there is an associated chronic peritonitis.

*Etiology.*—A history of repeated inflammations of one of the organs, usually the heart, is about all that can be cited as of possible etiologic significance. A number of cases have been found in which tuberculous foci existed in some region of the body. In several cases death was caused by an acute disseminated tuberculosis. Cantu (cited by Hager) explained the peculiar course on the supposition that a peculiar form of tubercle bacillus, probably avian, was active.

*Pathology.*—The most common lesion is the obliteration of the pericardial sac. In some cases marked calcification existed, indicating a postdegenerative process, as well as a proliferative one.

In most cases the pleura suffered a like change, though in a lesser degree. In nearly all there was an associated perihepatitis, and in many a marked increase of the intrahepatic connective tissue. The peritoneum is generally described as being thickened and opaque, with many adhesions. Kelly speaks of his case as presenting recent hemorrhagic peritonitis. The upper abdomen in the region of the liver is usually most intensely affected. Heide-

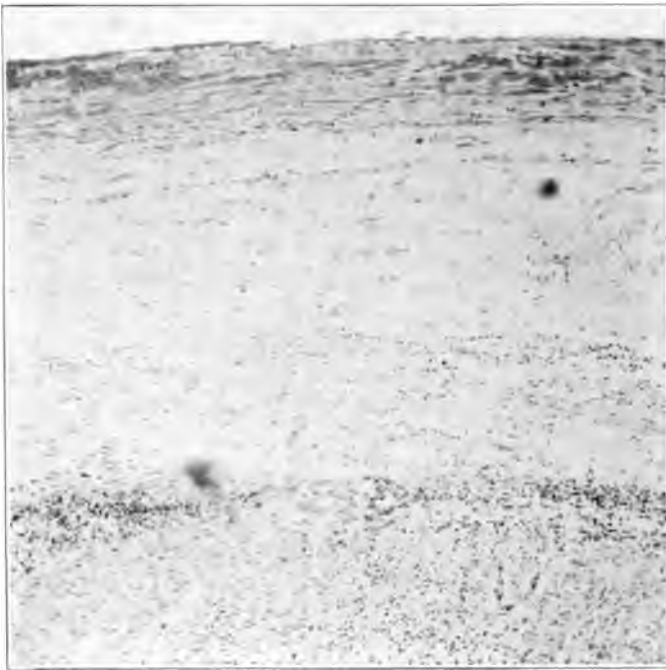


Fig. 210.—Chronic hyperplasia of the peritoneum. The upper three-fourths of the picture represents newly formed tissue. The fiber bundles do not respond specifically to any stain.

mann speaks of involvement, particularly of Douglas' pouch in his case.

In Hübeler's case, as well as in those of White, the peritoneum alone was involved. Their cases had much in common with certain forms of tuberculous peritonitis, except that the perihepatic peritoneum was intensely involved.

Kelly explains the associated lesions by assuming that there is a primary affection, of whatever nature it may be, beginning in the

peritoneal cavity, by virtue of the constant reaction of the current toward the diaphragm, and exciting its reaction here, subsequently extending to the pericardial and pleural cavities.

I have observed one case in which there was extensive thickening covering a large part of the area of the peritoneum. The pseudomembrane was several millimeters thick and was composed of poorly staining connective tissue fibers (Fig. 210). It appeared as if an exudate had formed which precipitated fibrillar fibrin and then for some reason the change to fully developed fibers was held up. I have seen the same thing in an imperfect state in very slowly developing cases of diffuse peritonitis. In the latter instance the development of the fibrils was still more imperfect.

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## CHAPTER XXVII

### THROMBOSIS AND EMBOLISM OF THE MESENTERIC VESSELS

Under this caption may be classified those abdominal disturbances which result from a primary occlusion of a mesenteric vessel, either from local disturbance (thrombosis) or by a foreign body transported from a distance (embolism). The essential feature of this disease is a necrosis of the gut wall secondary to occlusion of the mesenteric vessels. There has been a tendency, particularly by American writers, to include those conditions in which the mesenteric vessels become occluded secondary to some disease of the gut itself. The extent to which this confusion may lead can be appreciated when it is remembered that in most instances when there is necrosis of the gut wall there is some thrombosis of the vessels of the mesentery. Thus in the majority of necrotic appendices the vessels in the mesentery will be found thrombosed, if careful microscopic examination is made. Though the gangrene may be the result of the occlusion of the vessel, the vessel occlusion is the result of an inflammation of the appendix.

This condition is one of the rarer accidents of the abdomen. Because of a failure to separate the various types above noted, it is impossible to determine how many cases have been reported. Jackson, Porter and Quimby were able to collect 214 cases in 1904.

**Etiology.**—The causes which may lead to the closure of the vessels differ in the arteries and veins. Emboli affecting the arteries arise chiefly from endocarditis. This type is seen chiefly in young persons with primary endocarditis, or in older persons who develop clots after a period of uncompensated myocardial disease, as in cases reported by Kiliani in which clots had formed on the chorda tendinea, and Butlin in which a particle from a growth, evidently an old clot, had occluded the vessel. Thromboses are most often dependent on arteriosclerosis either as a part of a general process or a localized process due to a change in the

mesenteric vessels alone. These atheromatous deposits not alone destroy the elasticity of the vessels, but also narrow their lumen and roughen their intima. In some instances an atheromatous deposit may in itself be so great as to close the lumen of the vessel. Lorenz reports a case in which the finer branches of the superior mesenteric artery were obliterated by intimal proliferation, the end result of a multiple neuritis. When the obliteration from the endarteritis is not complete spasmodic contracture of the vessel wall is thought by some to be capable of completing the closure of the lumen.

In some cases a previous disease of the gut has existed. Gordon and Elliot reported a case in which a hernia had been strangulated some time previously. Höster had a patient who had suffered from diarrhea previous to the thrombosis.

Another group of cases followed operations involving large veins. Delatour had a thrombosis of the superior mesenteric vein following splenectomy, and Küster a like accident following pyloric resection.

Still others have followed unrelated diseases. McWeeney saw a patient in whom an abscess in the neck, followed by erysipelas, preceded the mesenteric thrombosis.

Enteritis, surgical infections, puerperal thrombosis of the pampiniform plexus, milk leg, phlebitis of the legs following typhoid fever and malaria have been noted as causes. Trauma, necessitating ligation of vessels, has been responsible and accidental ligation during surgical operations has brought about the conditions.

**Pathogenesis.**—Notwithstanding the rich blood supply of the intestine the occlusion of a vessel acts like the occlusion of an end artery. Many experiments have been made to determine the reason for this. The very complexity of the anastomosis seems in part to be responsible for the occurrence of blood stasis. Welch and Mall showed that the blood producing the infarction reaches the afflicted area by way of the anastomosing arteries and that the hemorrhage is the direct result of retardation of the blood flow. The ischemia they showed is due in part to spasmodic contraction of the muscle wall of the gut. Mall found that a gut 25 cm. long when quiescent shortens to 15 cm. during contraction and at the same time becomes ischemic. Mall has also shown that where the veins pass from the submucosa they have muscle walls so thick



that they resemble the arteries. These likely contract when the wall of the gut contracts.

I have sought to study this problem by injecting the mesenteric



Fig. 211.—A gut, the vessels of which have been previously injected with a solution of silver nitrate, is treaded on a test tube and covered with another to facilitate the study of the termination of the vessels. By revolving this under the objective of a binocular microscope the vessels can be accurately followed.

artery with a solution of silver nitrate. By threading a segment of gut over a test tube and covering it with a second tube, a circular slide is secured, (Fig. 211), the space between the walls of the two tubes being filled with glycerine. By placing a small electric bulb within the inner tube one can follow the vessels about the circumference of the gut. From these studies it seems to me that the chief factor is the failure of a collateral circulation to form, in that the supplying vessels are too small to supply the needed blood, just as if all the water for the inhabitants of a block had to come from a single inch pipe. If each pipe were supplied from a large main the supply would be adequate. The vessels are end arteries in the sense that they anastomose with their fellows coming around the gut from the opposite side. Deckart and Neutra have studied the cause of the absence of a collateral circulation. Cohnheim explained the absence of a collateral circulation because of a coincident closure of the anastomosing vessels. In a case described by Ponfick an actual occlusion of the smaller branches was demonstrated. Litten's experiments seemed to demonstrate that the vessels normally function as end arteries because a collateral circulation could be produced only by a higher pressure than occurs normally. Faber believed it was due to back pressure from the portal circulation. It would seem that the constriction of the gut by increasing the capillary resistance adds much toward making the formation of a collateral circulation more difficult. Rosenbach emphasizes the importance of this point.

That a collateral circulation to a degree is possible is shown by a case reported by Karcher. A case in which the superior mesenteric artery was plugged did not proceed to gangrene. He assumed that the area was supplied by the colica sinistra. In a case reported by Chiene both mesenteric arteries were occluded and the circulation was carried on by the superior hemorrhoidal and the colica sinistra and dextra. Cohn describes a case in which an area of the transverse colon had been affected and was cured by the establishment of a collateral circulation. Virchow describes a case in which the collateral circulation was established by branches of the pancreaticoduodenalis and inferior mesenteric arteries. Kaufmann describes a case in which there was a partly formed collateral circulation. The ileocolic artery was occluded but the part of the

colon supplied by it was unaffected (because the colon is less capable than the small intestine of contracting spasmodically). Robson reports that in a case of injury by puncture, the abdomen was opened and was found filled with blood. The superior mesenteric vein was found injured and was tied. Recovery followed. The colon in general is less susceptible than the small intestine to changes in the circulation. My notion of the reason for this is that the colon is less capable of constricting its vessels by violent muscular contraction. Roughton reported a case in which the *arteria intestini tenuis* was ruptured close to its origin from the mesenteric vessel and was ligated. Recovery followed.

**Pathology.**—Broadly speaking thrombotic and embolic processes in the mesentery may be divided into hemorrhagic infarction and anemic necrosis. This scheme helps to a better understanding in many cases both of the anatomic findings and the clinical manifestations. In a few cases pure forms are actually found in practice. Kader in his experiments showed that when the artery and vein were compressed but not completely occluded hemorrhagic infarction took place, but when complete occlusion was made anemic necrosis followed. This is quite in accord with the laws of the occlusion of vessels. Talke objects to the above theory on the basis of a case in which with simultaneous closure of the artery and vein hemorrhagic infarction took place. Similar cases were reported by Taylor and Groskurth, while in a case reported by Grawitz there was an anemic necrosis though the artery alone was occluded. The mesenteric vessels do not make up the entire picture. If there is an occlusion of the anastomosing plexus about the gut the fact that there are not open vessels above does not cause a hemorrhagic infarction. Also a hemorrhagic infarction, if the process is slow, may, after coagulation has taken place about the periphery, become an anemic necrosis after the hemoglobin has disappeared. In the localized forms, resulting from a local infective process, the area of infarction does not become hemorrhagic because of the fibrin thrown out by the primary inflammation. An exudation on the free surface of the peritoneum is produced by the same process that brings about the edema of the gut wall. The tissues even when blue-black retain their tinctorial reactions notwithstanding that the functional activity of the gut is destroyed. The toxicity of the

fluid exudate seems in no wise to influence the tinctorial properties of the tissues. The gut wall may form adhesions to the parietal peritoneum as a case reported by Watson showed. The process here is like that commonly observed in various cysts with twisted pedicles.

The end result of the occlusion is a necrobiosis of the gut wall. There is marked exudation into the walls of the gut primarily of serum, secondarily of blood cells. The process is really an ecchymosis as may sometimes be seen with the naked eye. The gut wall may be much thickened. A like process is seen in miniature in necrosis of the appendix when there is a thrombosis of the appendicular artery. When gangrene follows an area of the wall separates and a perforation results. This requires from seven to ten days.

**Symptoms.—Pain.**—Pain is the most common symptom. It is usually sudden in onset, diffuse in distribution, constant and dull in character with severe colicky exacerbations. In some cases the pain is slow in onset, due possibly to gradual closure of the lumen of the vessel. This is not always an adequate explanation, however, for in a case reported by Litten in which the pain was gradual in onset but which gave evidence postmortem of having become completely closed some days previously. The pain is due to violent colicky contractions. Borszékey and Kader think it is similar to intestinal claudication. Later in the disease peritoneal irritation comes in evidence. This course is particularly in evidence in those cases in which but a small segment of gut is involved. In such cases after the diffuse generalized pains subside, local symptoms dominate the field. In one case I observed after four days the pains became localized in the right iliac fossa. Several loops of the blackened gut, adherent to each other, occupied this region. The parietal peritoneum covering this site was intensely infected.

**Nausea and Vomiting.**—These symptoms are present as frequently as in other acute abdominal crises and bear nothing distinctive, except that later in the disease the vomited matter may be blood stained.

**Diarrhea.**—Diarrhea is the most distinctive sign and is present in the majority of cases. It is particularly significant when blood stained, as it frequently is. Diarrhea may precede the pain by some days as is shown by a case reported by Bradford. Jackson's

statistics show the presence of blood in the primary diarrhea in 19 per cent of the cases and at some time in the course of the disease in 41 per cent. Bloody stools are regarded as the most significant sign.

*Tenderness.*—Tenderness is usually present to some degree. It is usually generalized but later in the course of the disease when a limited segment of the gut is involved may become localized.

*Distention.*—Distention is usually present to some degree. Evidently the infiltration of the walls of the gut present the extreme distention seen in general peritonitis.

*Peritoneal Exudate.*—In harmony with the pathology of the disease the escape of a moderate amount of bloody serum greets the surgeon when the peritoneum is opened. The appearance of this fluid indicates the character of the lesion.

*General Symptoms.*—Some cases show marked increase of the leucocyte count. In some cases there was sugar in the urine. Temperature is usually below normal but sometimes it is elevated, particularly when there are complications, as endocarditis, peritonitis or toxic absorption. Puerperic spots have been noted by Talke and Osler.

*Diagnosis.*—Sudden abdominal pain, particularly in persons with cardiac disease should suggest the possibility of mesenteric thrombosis. If a bloody diarrhea supervenes the diagnosis becomes a probability. Usually the diagnosis has been made after the abdomen is opened. The escape of blood-tinged serum indicates the nature of the trouble, and the surgeon has but to seek its source. Tissues giving rise to such fluid have a peculiar odor, as is most often observed in ovarian cysts with twisted pedicles. The blue-black gut tells the tale once it is brought into view.

*Treatment.*—Because of the extent of gut involved, treatment has rarely been successful. The affected portion of the gut, if it is not too extensive, may be drawn out of the abdomen and an opening made. The affected segment has been removed with success in several cases.

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## CHAPTER XXVIII

### DISEASES AND INJURIES OF THE GREAT OMENTUM

#### Inflammatory Tumors of the Omentum

**Definition.**—Under this heading are included those conditions of chronic induration of a part of the omentum in which a tumor-like mass is formed by cellular and fibrinoid infiltration of the tissues. It has analogues in other regions of the body, notably in chronic salpingitis, in the woody phlegmons of the neck, etc. In appearance it represents a border line between reactive processes and malignant tumors. It is due to a peculiar low grade of infection and is characterized by a fibrinoid degeneration of the connective tissues and an infiltration of polynuclear and mononuclear cells. Hölzländer suggested the descriptive name of *epiploitis plastica*.

The literature deals largely with those cases which follow resection of the omentum, particularly following hernia operations, and no doubt the most typical cases have followed such operations. Not infrequently, however, the omentum becomes inflamed because of torsion or from strangulation in an irreducible hernia. When the reaction is great pronounced disturbances result. Lesser degrees most likely occur which are never recognized. In many cases in which the patient does not recover normally after an operation involving ligation of the great omentum, if careful search is made some degree of this condition may be discovered. When the omentum becomes attached to a variety of inflamed lesions it may undergo similar but much less marked changes. This is not likely to occur in low grades of infections of the appendix and cecum, gall bladder, and Fallopian tubes. This condition must be distinguished from the usual thickening of the omentum common to all infections.

**History.**—The literature bearing on this subject is all of relatively recent date. Braun was the first to call specific attention to this condition. He discusses his own cases and reviews thirty cases



from the literature. Zesas and Holländer bring the literature to near the present date.

**Pathogenesis.**—The exciting factor is nearly always situated in some neighboring organ, notably appendicitis, salpingitis, strangulated hernias, and particularly following operations for hernia in those cases in which portions of the great omentum were ligated with silk. The essential factor is chronicity. Usually some time after the infection the omentum begins to enlarge. This enlargement continues for a period usually from three to six months, then regression begins. Roughly speaking, about nine months are required for the completion of the cycle. One case has been reported (Boeckel) in which the induration did not begin until three years after the operation that occasioned its development. On the other hand, an acute development may take place, simulating a general peritonitis, as in a case reported by Pantzer. When the onset is acute there is normally an attendant venous thrombosis due to trauma or torsion. There seems to be no specific organism. A coccus is usually recovered. It is probable that the virulence rather than the variety of the organism is the determining factor.

Two avenues of infection are possible: From direct contact with an inflamed organ and by infection carried from a distance, as Walther suggests. Prutz and Monnier doubt the existence of the latter type. There is no case recorded that would require such an explanation. It seems more plausible that bacteria free in the peritoneal fluid may be taken up by the omentum or that owing to some disaster, as torsion, a primary affection of the organ in question is produced because of the nutritive disturbance.

That form which follows operation usually follows some fault of technic if the parts were healthy at the time of operation. An astonishingly large number have followed the use of silk in the ligation of the omentum. In these cases it is to be presumed that the infection was carried with the silk.

The question as to whether the condition can arise without infection is answered in the affirmative by Lucas-Championnière. This view seems tenable. Simon reports a condition arising slowly during a hernia without evident infection. A hemorrhagic exudate due to toxic or mechanical causes under certain conditions seems capable of producing it. This condition may be produced by

torsion when the torsion is not great enough to produce a gangrene. I have been able to produce a very good imitation of this condition by placing within the folds of the great omentum a pledget of cotton soaked in a culture of pus organisms, or in blood that had been heated to 60°.

A condition very much like the one under discussion sometimes occurs in the abdominal wall, particularly in the region of the inguinal canal, following an operation for hernia, and in the gut wall in certain chronic conditions, as chronic appendicitis, or cholecystitis.

**Pathology.**—The essential factor is chronicity. The cellular element is made up largely of polynuclear leucocytes in the more acute cases, while in the more chronic forms there may be very few polynuclears, and large round cells with ovoid vesicular nuclei abound. The tumor mass may be made up largely of cells or the bulk of it may consist of edematous fibrinous tissue. In either instance the connective tissue loses its specific reaction to acid dyes and approaches the fibrin reaction, staining a brownish yellow with picrofuchsin. Within the meshes of this connective tissue may be a granular fibrin and often red cells in a fair degree of preservation. In the more cellular variety the groundwork is made up of connective tissue of this kind and in its meshes cells of the varieties above named find lodgment.

Variations from this type may occur in the direction of destruction of tissue, even to actual abscess formation, or on the contrary an attempt at organization, characterized by the presence of spindle-form cells simulating young connective tissue cells may be present. These various conditions represent the different degrees of activity of the irritative process. It seems likely that this condition represents a chemical rather than a bacterial process, being due to a fermentative process. In some cases apparently there is an infection. This is particularly true in those arising from an acutely inflamed organ. In thrombosis following ligation the whole process may run its course without the intervention of bacteria.

**Symptoms.**—The onset may be relatively acute for a condition destined to such a chronic course. Diarrhea, pain, and fever may be present in the beginning. On the other hand general malaise may be the only symptom until the tumor is discovered. It is us-

ually more or less globular. The most common site is between the umbilicus and the pubes, although sometimes, as in Holländer's case, the tumor may be in the right groin. They vary in size from a fist to an adult head. I once saw a case that filled the entire abdomen below the umbilicus. The omentum formed a mass 10 cm. thick and 25 cm. in diameter.

When a considerable portion of the omentum has been removed, the tumor may be situated near the transverse colon. I observed such a location where half the omentum had been removed with a myoma to which it was adherent. On the other hand, the tumor may be located low in the abdomen. In omenta little or none at all of which has been resected, particularly those that have dwelt in hernial sacs, the tumor is apt to be low in the flank, even in the pelvis.

The mobility varies within wide limits. Usually it is fixed in position, often near the line of incision, if an operation has preceded the development of the tumor.

The density of these masses is usually marked, being in most instances dense and elastic, and in some cases quite equal to the density of carcinomata. Generally speaking the mass is uniform and is free from the small bosselations which characterize carcinomata.

**Diagnosis.**—The presence of a tumor in the region of an operation which involved the great omentum may lead one to suspect the presence of such a condition. The probability is heightened if there are indefinite digestive symptoms, slight pain, a little fever, and a moderate leucocytosis. In rare instances the leucocytosis may be high. One of my cases had a white count of twenty-five thousand, 90 per cent of which were polynuclears. The difficulty in some cases is in determining if the disturbance may not be due to some other cause. The case just quoted followed resection of the omentum with hysterectomy for myoma. There may have been some trouble with the wound in the pelvis, though no disturbance could be demonstrated. That it was due to the omental tumor is rendered likely because the height of the disturbance was not reached for six weeks following the operation. When the mass is large enough to press on a segment of gut, symptoms of obstruction may appear. More pronounced disturbances may take place, even to complete obstruction when the mass surrounds the gut.

Enterostomy was required in Schmieden's case and in one of my own cases. Colicky pains and vomiting do not always mean obstruction, however. Becker proved in his case by a bismuth meal that no obstruction existed. The type that develops independently of a surgical operation is usually mistaken for a malignant tumor. Because of the relatively rapid onset and size of the mass, sarcoma is most often diagnosed, while if small and they cause some constriction of the gut, carcinoma is most apt to be diagnosticated, particularly if the mass is in the epigastric region. I have twice made this error. Not infrequently an exploratory operation is done and the abdomen is closed and the patient recovers despite the grave prognosis. If after exploratory operation a malignant condition is diagnosticated which later recovers, this type of trouble most likely was present. As a general rule if, when the abdomen is opened, the mass is found adherent to the abdominal wall, it is inflammatory and not neoplastic in character. If doubt exists and the tumor is cut into, it will be found to be pale pink in color and exudes fluid. In some cases the final outcome must decide the character of the tumor.

A microscopic differentiation may be possible. When a preponderance of polymorphic leucocytes is present a reactive process may be diagnosticated without question. If the large mononuclears alone are present the resemblance to a sarcoma is sometimes very close indeed. When the process involves a gut wall the differentiation from sarcoma may sometimes be particularly difficult. It seems more than likely that in some instances the process may begin as a reactive one and end as a neoplastic one. When the process takes place in the abdominal wall the resemblance to a desmoid may be very close in the more chronic types. The presence of any polynuclears at all speaks against desmoids. In fact it is likely that the desmoids represent the end product of just such an inflammation. In all instances the clinical history must be taken into account.

**Treatment.**—Reynier advised the removal of the mass just as though it were a malignant tumor. This is not such an innocent procedure unless the mass is free from adhesions, except for its omental attachments. Prutz had three deaths in 9 cases operated. Braun removed a wedge of tissue to hasten regression. The gen-

eral tendency is one of regression, though many months may be consumed in the process, and Enderlen is certainly right when he advises a policy of waiting. This author advises the use of hot air and wet packs. Simple incision into the tumor hastens resolution by allowing serous drainage. Drainage of pus usually follows such a procedure and it is not certain whether it is caused by bacteria existent within the tumor or introduced from without after the incision has been made. Apparently bacteria within the tissue, securing more favorable conditions for development, set up the suppurative process. This seems likely because in a goodly number of recorded cases in which incision had been made the extrusion of silk ligatures followed.

Not infrequently suppuration takes place without incision, even many months after the beginning of the disease, demanding incision. Such cases may be detected because of the greater pain and tenderness with the increase in leucocytosis which attends them. The area breaking down is usually too deeply seated to admit the detection of fluctuation. Because some of them break down Prutz recommends that all cases be treated as intraabdominal abscesses. Abscesses must be found before they can be opened, however, and usually the foci of liquefaction are small and numerous, as in some lung infections, and the general area of maximum involvement only can be drained. In large masses the site of the trouble may not be located with certainty. In such instances the center of the area may be boldly sought provided this does not lead in the direction of a hollow viscus. Failure to recognize this caution cost Schmieden a gut fistula. This plan is too radical as a general principle, for most of the masses resolve if allowed sufficient time, and active interference is advisable only when there is evidence of suppuration.

Sometimes the fecal circulation may be interfered with, particularly in the transverse colon, and operative interference may be demanded on that account. Partial removal of the mass may meet this indication as recorded by Monad. Interference with the fecal current is less likely to occur when the omental hyperplasia is primary than when the infection reaches it via a hollow organ, particularly the appendix. In some cases enterostomy or anastomosis between the obstructed loop and the colon may be required as

in Schmieden's case. Such a procedure is better than to remove the entire mass.

**Defensive Reactions of the Omentum.**—The general capacity of the omentum to react to irritation has been considered in the chapter on the formation of adhesions, and its specific action receives

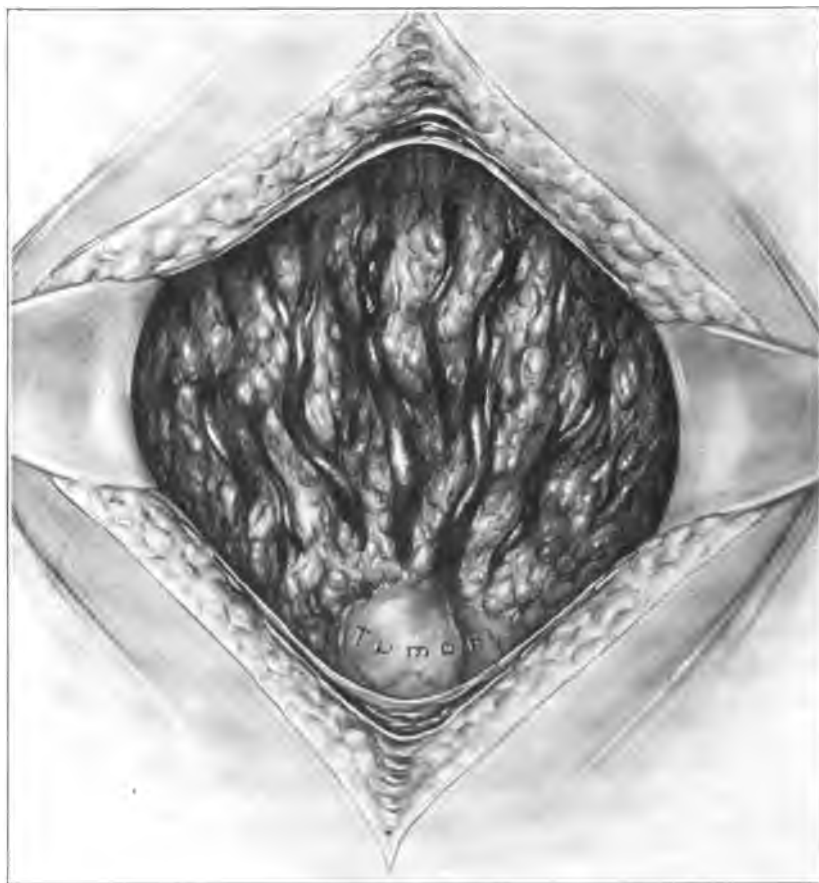


Fig. 212.—Attachment of the omentum to a myoma that had suffered a disturbance of nutrition. Note the pronounced dilatation of the veins.

mention in separate chapters in which it performs its function. Under this heading are gathered a number of interesting conditions which do not lend themselves for a more specific discussion.

**The Omentum as an Accessory Source of Nutrition.**—When an

organ becomes distressed because of some accident to its blood supply the omentum is able to attach itself and add to the supply, or what is equally as important, conduct away the stagnant venous blood. This is best illustrated in those cases where a pelvic tumor, because of some interference with its blood supply, has its nutrition disturbed. Here the omentum often becomes attached (Fig. 212) and aids in reestablishing an efficient circulation. The great capacity of the omentum, to render such aid has been shown experimentally. If all the vessels of the spleen are ligated the omentum may envelop and prevent gangrene.

This capacity of the omentum to aid venous return is made use of in cases where the return circulation of the abdomen is hindered by a cirrhotic liver. It was hoped that by attaching the omentum to the abdominal wall a sufficient accessory circulation could be established and thus circumvent the liver. The task is of course too great, but it is paying a delicate compliment to a very versatile organ.

**Fat Necrosis.**—This condition represents chiefly a degeneration, and only secondarily a necrosis. Whenever any lesion of the pancreas permits the escape of the fat-splitting ferments, areas of the fat with which it comes in contact become necrotic. The omentum being the organ most frequently attacked, it receives most attention. The areas so affected appear as small mass areas of dull white set into the surrounding omental fat. The line between the affected and non-affected areas is very sharp. The necrotic areas vary in size from a mere point to large patches (Fig. 213). On section these necrotic areas show the fat cell to have undergone a granular degeneration (Fig. 214). The nuclei as well as the protoplasm undergo this change. After the condition exists for some days the unaffected area undergoes a reactive round-celled infiltration.

### Torsion of the Great Omentum

**Definition.**—By this term we mean the rotation of the great omentum on its longitudinal axis of sufficient degree to produce disturbance of the circulation. Usually the entire omentum is involved but a portion only may be affected. Occasionally instead of twisting on its long axis some other axis is involved.

**History.**—Rudolf and Vignard and Giraudeau have presented



Fig. 213.—Fat necrosis in the upper part of the omentum from a case of necrosis of the pancreas. The white necrotic areas are set into the unchanged fat of the great omentum like tiles in a floor.

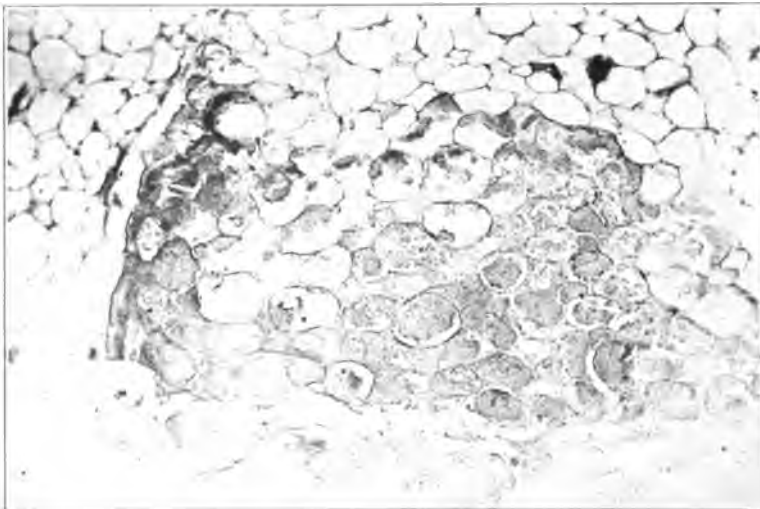


Fig. 214.—Fat necrosis of the omentum in a case of acute pancreatitis. Note the opaque degeneration of the fat cells. Some of these have moderate cellular infiltrations about them.



statistical papers. Hadda collected 92 cases and v. Cackovie collected 94 cases and added 4 new ones. Prutz and Monnier bring the total recorded cases to 134. The literature abounds in individual case reports not included in this summary.

The possible types are manifold, as indicated by the classification proposed by Prutz and Monnier. The practical requirements are met by a more simple classification, however. The most frequent variety is that associated with a hernia, next in frequency is the type in which the omentum lies free or at least wholly within the abdominal cavity, and finally the small group which is complicated by the presence of some other disease either of the membrane itself, such as a primary or metastatic tumor of the omentum or the presence of some other disease to which the omentum may be attached, as an ovarian cyst.

**Pathogenesis.**—It is difficult to determine in many instances what anatomic changes existed before torsion took place and what changes followed the incarceration. That increases in bulk of the omentum, particularly of an isolated segment of it, may act as a predisposing factor may be assumed from the fact that other tumors of like bulk undergo torsion. Torsion in a considerable proportion of cases is found in corpulent persons which in itself suggests a primary bulky organ. An unduly fat omentum presents the same mechanical problems that an ovarian cyst does. Gynecologists have speculated much on the mechanics of torsion which is of interest here, and we are enabled to discourse learnedly at the expense of their labors. Aside from the bulk of the organ an uncommon elongation of the organ or a segment of it has been observed. This is true naturally of those cases in which the omentum is found in a hernial sac. It must have been unusually long originally or it could not have become an inhabitant of a hernial sac extending into the scrotum, but its sojourn in the sac no doubt tends to lengthen it still more.

In order to determine the agent active in producing the rotation the intraabdominal and the herniated varieties must be considered separately. The intraabdominal, as already indicated, may be explained the same way as the torsion of ovarian cysts. As the body turns the upper border of the tumor tends to travel more rapidly than the lower just as the upper segment of a wheel travels

more rapidly than that segment in contact with the ground. The action of the abdominal muscles and gravity both may act in exaggerating the rapidity of the movement of the upper segment. The action of the abdominal muscles may act directly on a segment of the tumor, not only by increasing the intraabdominal pressure as Hadda thought. The increase of intraabdominal pressure might be a very potent force if it involved chiefly one portion of the abdomen, but if pressure is increased in all directions movement is not imparted to the tumor. Bakes emphasized the influence of position on the movements of the omentum as observed during operations in the Trendelenburg position. As Prutz and Monnier point out dislocation due to changes of position could explain rotations to  $180^\circ$  only while the torsions as usually observed represent at least  $360^\circ$ , sometimes much more, even to eight complete revolutions. We might assume that torsion can be produced by successive turns of half a circle.

My own studies on the movements of the great omentum lead me to believe that the omentum is not such a helpless structure as might be supposed. Though immobile it does change its position; of that there is no doubt. The cause of this movement will be more fully gone into elsewhere. Here suffice it to say that when a foreign body causes a marked hyperemia with exudation in that portion of the omentum in contact with the foreign body, cells are attracted. There is a possibility that this attraction may carry with it the web-like omentum. One might liken the omentum to a May pole. The ribbons represent the tissues of the omentum and the children the leucocytes. In their gyrations the children carry the ribbons with them. I should not for a moment argue that the large masses one sees after torsion has taken place are capable of movement, but the omentum may roll about as one sees it do about a pledget of gauze and subsequently acquire its bulk due to a disturbance of the circulation consequent to the rotation. This would explain those cases only in which there is torsion in a normally thin omentum. Large fatty masses could hardly act so.

The most common variety of torsion is that affecting omenta the inhabitants of hernial sacs. Steiner in 62 collected cases found only 7 not associated with hernia and Hedley in 73 cases all but 13 were

associated with hernia. Three of my 4 cases were in association with inguinal hernias.

The elongated omentum residing in the hernial sac becomes clubbed and often there is a more or less sclerotic constriction of the omentum near the inner abdominal ring. This may often be noted in irreducible omental hernias without torsion. My first case suggested to me that the curve of the inguinal canal carried the clumped mass of the omentum like the "lands" of a rifle carry the bullet. I once operated on a male aged 36 with a huge inguinal hernia in which not only the omentum, but the cecum as well, which also occupied the sac, had undergone torsion. Hadda has since thought the same way. Quénue has called attention to the fact that in proportion to the number of omental hernias, torsions are rarities. Pressure of a truss or effort at reduction likewise fails to explain the accident.

**Pathology.**—When the torsion is complete gangrene of the parts below the stricture must occur. However, obstruction usually is not complete (Fig. 215) and the omentum obtains some nutriment from the surfaces to which it becomes attached. In this regard it is exactly a parallel with the walls of ovarian cysts which have become twisted.

In the degree with which reaction is possible cellular activity may be present. Usually many red cells have become extravasated and but few leucocytes are present while the connective tissue loses its specific staining reaction. When nutrition is reestablished either through the natural channels or from secondary attachments leucocytes and round cells may be present in abundance and the connective tissue show a fibrinoid rather than a narcotic change.

**Symptoms.**—There is usually a preexisting hernia, perhaps of many years' duration. The actual torsion usually follows some violent effort or prolonged bodily activity. Sometimes there are slight attacks of pain. These may persist for some days or even weeks. In a number of cases recorded lesser signs extended over a period of many months. Then come the more pronounced symptoms of pain in the region of the torsioned omentum. Nauseas and vomiting and sometimes collapse follow. One of my patients dropped in the road and lay writhing in pain until transported to the hospital by passers-by. There may be radiating pains either to-

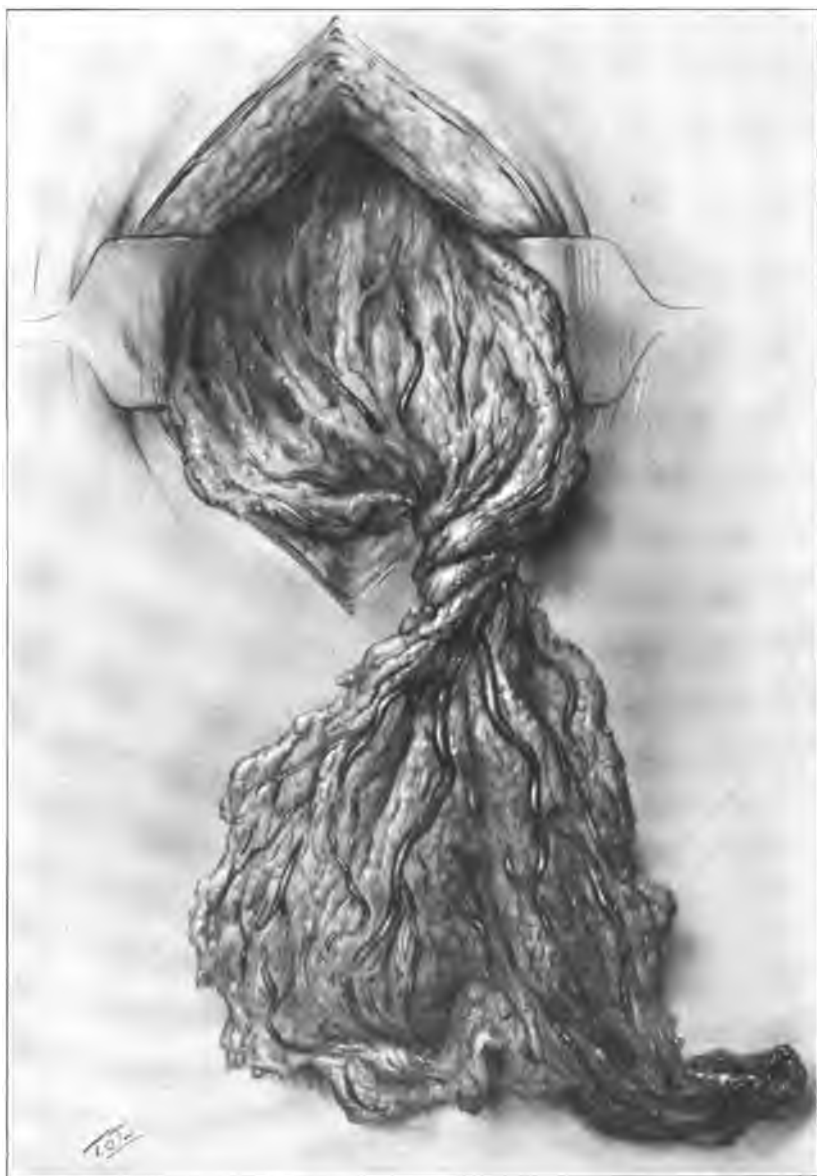


Fig. 215.—Torsion of the great omentum. The portion below the twist was black and dense while that above was unchanged save for the marked dilatation of the veins.

ward the side or down the leg. If a hernia has been present it likely becomes irreducible, much enlarged and invariably very sensitive to manipulation. With this phenomenon prominently before him the surgeon will hardly escape the diagnosis of a strangulated hernia, if the onset is violent, or of irreducible hernia if the onset is more gradual. I have made both errors. In my first case the patient long carried a large irreducible left inguinal hernia. He bore this affliction because of a chronic nephritis. He was suddenly seized with a pain in the hernia after an unusual activity in invoicing a stock of clothing. He presented a large hernia which was very tender to the touch and the skin over it was edematous. There was an indurated mass extending upward and medially from the inguinal region. This mass puzzled me but I did not grasp its significance. The inguinal canal was laid bare under local anesthesia and a black omental mass was disclosed. The hernia contained no intestine. As the blackened mass was dislocated from the wound it was found to extend upward into the abdomen and the ridge above noted was found to be due to the black edematous omentum. Not until this was withdrawn and a twisted area below the transverse colon was brought into view did the nature of the condition dawn upon me. The torsion was from right to left and amounted to full 360 degrees. The mass was removed just above the point of torsion. The separation of the mass from the hernial sac caused pain as did the traction necessary to bring the upper end of the mass into view. The patient showed distinct exhaustion and vomited several times during the first day following operation, but recovery was uneventful. My second error was made in the case of a right-sided hernia in a man of seventy-six. He had had a hernia for fifty years, but until recent years was retained by a truss. When the truss refused to retain the mass he discarded it and allowed the hernial mass to come down at will. I examined him several times and a reducible omental mass could easily be made out, but could be reduced at will, and it returned spontaneously into the abdomen at night. As he became more feeble the mass became larger and refused to retract into its normal habitat as of yore when the patient assumed the recumbent position. The mass became tender from time to time but a period of recumbency usually restored him to relative comfort though the mass did not return into the abdomen.

Without known cause the pain continued to increase and caused a constant pain down the anterior crural nerve. The hernial mass increased in diameter and became more tender and the patient vomited. There was an indefinite induration extending upward in the direction of the deep epigastric vessels. Despite a similar observation in the case just mentioned the significance was not grasped and a diagnosis of strangulated omental hernia was made. Exposure of the inguinal canal showed a large club-shaped omental mass which was blue-black in color. As it was drawn downward a twisted stalk was disclosed. Some of the gyrations seemed to be old and were fixed by fibrous adhesions. In fact it seemed as though all the turns antedated the onset of the acute symptoms. It gave the impression as though the torsion had occurred without producing marked symptoms and that it was only a later associated thrombosis that caused disturbance sufficiently great to seriously disturb the nutrition of the mass. The torsion in this case was from left to right. The number of turns could not be definitely made out.

The only sign of unusual character observed in these two cases was the induration extending upward from the inguinal canal. This might be of some service in diagnosis, but a like condition is observed in mesenteric thrombosis without torsion.

I operated upon a colleague recently who had had an irreducible inguinal hernia for many years. When the inguinal canal and scrotum were opened a mass as large as a fist was dislocated from its bed. At the lateral inguinal ring the omental stalk was less than an inch in diameter. The vessels described a spiral about the narrowed place. It was evident that they could have assumed this position only by the twisting of the lower extremity of the omental mass. The clubbed portion occupying the scrotum was removed only as a matter of mechanical convenience. This mass was firmly adherent to the hernial sac beginning at the level of the pubic spine and extending downward for 15 cm. Possibly the circulation was disturbed at some time giving rise to these adhesions. From these cases it seems there may not always be a sharp dividing line between irreducible hernia and torsion of the omentum.

In the internal variety the general symptoms are those of some pronounced abdominal crisis. The absence of a hernia makes the

nature of the disorder doubly hard to anticipate. It is only after the abdomen is opened that the nature is likely to be suspected. I once observed a condition of this sort associated with a more pronounced lesion. The patient, 46 years of age, gave a history of several severe abdominal crises. The most severe of these took place 15 months before she presented herself for examination. She had a temperature for three weeks following that attack, her physician said. When I saw her she had a renewed attack of the pain, though less severe than the previous one. A rounded mass, tender to touch, filled the pelvis and could be palpated above the pubes particularly to the left. A diagnosis of cyst with twisted pedicle was made. At operation as the cyst was delivered the omentum followed. The cyst was blue-black and the veins were filled with thrombi. As the omentum was drawn down there appeared a twist above which the omentum was normal though much congested (Fig. 215). The torsion was fresh and not over 180 degrees. The pedicle of the cyst showed the remains of an old twist and a new one. It would seem that following a partial twist of the ovarian cyst a year before, the omentum became attached and as the cyst underwent a renewed gyration it was compelled to follow. It is doubtful whether the degree of torsion the omentum suffered would have been sufficient to disturb the circulation had it not been attached to the seminecrotic cyst wall.

**Diagnosis.**—From the foregoing it is evident that when an omental hernia produces marked symptoms torsion of the omentum must be thought of. From my own experience I would suggest that torsion may be distinguished from simple omental strangulation by the fact that the latter produces a tumefied mass extending upwards beyond the internal ring, while the latter produces its disturbance within the confines of the inguinal canal.

In the variety contained entirely within the peritoneal cavity the surgeon may be content if he diagnosticates a surgical lesion. Prutz and Monnier note that a cursory examination during laparotomy may not be sufficient to discover a torsion and a nonoffending organ such as the appendix may be removed and the chief cause of the trouble, the twisted omentum, may be overlooked. This is merely another case in which if the surgeon does not know how

much of a lesion is required to produce a given group of symptoms, he is likely to overlook the real pathologic condition.

The chief lesion that is likely to be confused with torsion of the omentum is inflammatory tumors of the omentum. The chief difference lies in the fact that in the latter the onset is slow and gradually progressive, and usually follows some operation on the omentum, while in torsion the beginning is stormy and most often associated with hernia. In the internal variety of torsion and the omental inflammations not following operation the differentiation may be aided by considering possible etiologic conditions but in such cases direct inspection by laparotomy is better than hypothea-tion.

**Prognosis.**—Judging from the ability of an ovarian cyst to recover after torsion of its pedicle it is more than likely that torsion of the omentum is not incompatible with continued life of the patient. This is made probable because a careful examination of thick omental masses sometimes shows evidence of such a catastrophe in times past.

**Treatment.**—When a twisted omentum is discovered at operation, nothing remains but to sever the diseased portion and remove it. The appearance of the omentum above the twisted part is similar to that observed when it is attached to a pelvic tumor and the treatment is the same: careful ligation in several segments with ligatures large enough in diameter that they will not cut the thin-walled veins.

### Injuries to the Omentum and Mesentery

Generally speaking, traumatism of the omentum and mesentery is overshadowed by perforation of the hollow organs and rupture of the solid organs. It is usually only when injury of vessels occurs that rupture of the omentum and mesentery assumes importance. Rupture of the nonvascular areas it is true may furnish atria for subsequent hernias, but these are of very rare occurrence. Tearing loose of the mesentery from the gut for great distances may jeopardize the nutrition of the latter. This likewise is a rare accident.

**Etiology.**—Two general classes of injury may be defined; those due to blunt trauma and those due to penetrating wounds.



**Blunt Trauma.**—Thirty-four cases have been recorded. The most common cause was being run over by a vehicle, with 9 cases; next, pinching between two objects as between the bumpers of railway cars, or other vehicles with four cases. Kicks from a horse were responsible in two cases. Injuries from contact with the ends of objects such as a wagon tongue were responsible for five cases, and, finally, falls upon the abdomen were responsible for three cases.

**Penetrating Wounds.**—A bare half dozen cases have been reported as such. The paucity of these may be accounted for by the fact that generally other organs were injured at the same time and these overshadowed the mesenteric injury in importance. Obviously many more cases occur than is apparent from a reading of the literature. Severance of a mesenteric vessel, particularly from gunshot wounds, is not such a great rarity.

**Pathogenesis.**—The rupture of the mesentery in general may be said to be due to a force acting on the organs to which it is attached. For instance when an intestine filled with fluid, in response to pressure from above, travels downward with a greater force than the mesentery can withstand, it leads to rupture. Likewise if a force acts on the mesentery directly, if the range of its elasticity is exceeded, rupture must occur.

Usually the history of recorded cases does not admit of such accurate classification. Generally a trauma acts on the abdominal wall and at autopsy or at operation a rupture of the mesentery is found to have taken place. Thus Neumann reports a case of Garre's in which the mesentery was torn from the intestine for 150 cm. as the result of a blunt trauma.

The mechanism of wounds of penetrating objects requires no discussion.

**Symptoms.**—Usually there are surface indications of a blunt injury such as excoriations and contusions. The history may suggest the possibility of such injury if objective evidence is wanting, such as being run over by a vehicle or being struck by some object, as reported by Riegner and Steinthal.

Pain is present in degree of associated injury of the abdominal wall, or of hemorrhage into the tissues of the abdomen. A hematoma in the root of the mesentery, or a blood clot may irritate the peritoneum, as noted by Wilms.

The most constant evidence of serious injury is manifest by pallor, small and rapid pulse due to hemorrhage. This may be due to the injury itself or to the associated hemorrhage. Primary shock may be so great that the hemorrhage may be delayed, as shown by the case reported by Matthes. In such cases hemorrhage may start again as soon as the shock is recovered from.

*Hemorrhage.*—Aside from surface changes and character of the pulse the local accumulation of blood can often be detected by percussion.

*Diagnosis.*—The exact determination of an omental or mesenteric injury is hardly possible. The most that can be done is to determine the presence of an intraabdominal injury (see rupture of the intestines). In some evidence of pallor is present and the demonstration of blood in the abdominal cavity is possible.

*Prognosis.*—The prognosis is as varied as the type of injury. Primary shock may cause death or death may come from hemorrhage. If the hemorrhage is moderate spontaneous recovery with absorption of the blood is possible.

*Treatment.*—If evidence of grave abdominal injury is present operation is indicated. In the presence of marked shock the question arises, however, if operation should be undertaken at once or if the passing of the depressed state should be awaited. In the presence of profound shock with no evidence of intraabdominal hemorrhage, the appearance of a reaction may apprehensively be awaited. At best, procedure in all these cases is an exploratory operation.

When a ruptured mesentery is found the question of the vascular injury is the paramount one. If there is but an injury of the mesentery without vascular injury the cleft is closed by suture as a preventive against future intestinal obstruction. If a vessel of any considerable size has been ruptured the extent of gut supplied by this vessel must be accurately determined. If the detachment of the mesentery from the gut is greater than four inches or a vessel supplying a greater extent than this is injured intestinal resection must be made. The circulatory disturbance in the gut wall gives evidence of the extent of resection demanded as shown by the cases of Autenrieth and Reinicke. Too great a segment of

gut rather than too little had best be sacrificed in case of doubt, as shown by Lockwood's case.

Several experimental studies have been made to determine the value of reinforcing portions of intestine, the circulation of which has been jeopardized, by enveloping it in a fold of the great omentum. Benker found that the gut can be notably reinforced by these means. Scudder placed the omentum in the opening in the mesentery. Rosenstein resected a portion of the omentum and stitched it about the jeopardized segment of the gut. The result saved a gut freed from its mesentery for 20 cm. This author overlooks the fact that the mesentery can safely be severed for a greater extent from an inflamed than from a normal gut.

Isolated injury of large vessels has been reported in a few instances. Hagen reports the injury of the superior mesenteric vein; Mayo-Robson (1897) reports a similar case. I saw one in which the superior mesenteric artery was cut off by a rifle bullet.

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## CHAPTER XXIX

### TUMORS OF THE PERITONEUM

The peritoneum itself is the source of origin of relatively few true primary tumors. These of necessity must consist of endothelial or fibrous tumors. However, there are many tumors so closely associated with the peritoneum that practical considerations demand their study. The more common are the retention and proliferative cysts, usually within the layers of the mesentery or omentum. Many of these arise in vestigial structures not directly related to the component parts of the peritoneum. Small adhesion cysts, the result of some past inflammation, are common but have no clinical significance. The blood, and particularly the lymph vessels sometimes make distinct tumors. External to the parietal peritoneum are many solid tumors, the product of the retroperitoneal connective tissues, and teratoid tumors the result of embryonal displacements. Parasitic diseases may find lodgment, producing conditions closely simulating true tumors. The most common tumors of the peritoneum are the metastatic ones, developing secondarily to malignancies in some abdominal viscus.

**Classification.**—Considered in its broad sense, therefore, the tumor problem as it relates to the peritoneum must be approached in a thoroughly comprehensive manner. Henschen has proposed a classification possessed of many desirable features so far as it is related to the cystic conditions. This outline will, with some modifications, be followed here. The solid tumors as well as the metastatic ones may be arranged under the forms usually followed in the discussion of tumors. The following outline, therefore, may be suggested.

#### CYSTS.

##### A. Proliferative (True cysts).

1. Cavernous lymphangiomata.
  - (a) with serous contents.
  - (b) with chylous contents.
2. Cystic endotheliomata.
3. Dermoid cysts.

**B. Foreign Body (Infective) Cysts.**

1. Echinococcus.
2. Gas cysts due to bacteria.
3. Cysticercus cellulosæ.

**C. Retention (Secondary) Cysts.**

1. Blood cysts resulting from hemorrhages.
2. Adhesion cysts formed by the accumulation of exudate in pockets.
3. Degeneration cysts.
4. Hydropic cysts.

**SOLID TUMORS.**

1. Lipomata.
2. Fibromata.
3. Endotheliomata.
4. Sarcomata.

**METASTATIC TUMORS.**

1. Carcinomata.
2. Sarcomata.
3. Pseudomyxomata.

Such a scheme must present some evidence of incompleteness. This must of necessity be so since the genesis of some of the lesions are as yet not definitely established. To make this less apparent the tumors of the mesentery and omentum are discussed separately.

**Lymphatic Cysts of the Mesentery**

The justification for classifying these cysts as proliferative cysts is found in the structure of their walls. They are composed of connective tissue interspersed with many elastic fibers and usually an abundance of smooth muscle fibers in which are imbedded masses of lymphatic tissue. In the wall are usually found small clefts or cysts. The cell content is variable. Usually numerous cells surround the vessels. The free surface is covered with large endothelial cells that may appear arranged in several layers. The apparent stratification I believe is due to exfoliative processes. Klemm believed that they represented foreign body giant cells.

**Frequency.**—Berger collected 137 cases of these, three of which were in the mesocolon.

**Size.**—The size varies between that of an egg or less to the case reported by Weichselbaum, which represented two enormous cysts measuring 23x29x76 cm. each.

Multiple cysts have been reported, for instance one by Tuffier and Bennecke.

**Sex.**—Gildermeister in collected statistics of 44 cases found 26 in females and 18 in males. Braquehay found 4 females to 1 male.

**Age.**—Early adult life is most frequently affected. Gildermeister found but three recorded cases over 50 years of age, while 28 were under 30.

**Pathogenesis.**—The structure of these tumors is altogether homologous with the lymph cysts of the neck and axilla, and I may say also like the polycystic kidneys. It seems fair to assume that they have a like genesis. The formation is obviously laid in a congenital abnormality, but the process is capable of neoplastic development. This theory of genesis excludes the necessity of assuming that they are derivatives of degenerated lymph glands in order to account for the presence of lymph nodules. The congenital character of the anlage is further attested to by the fact that these cysts are usually observed in early life, as noted by Henschen and Klemm. How these arise in early life is explained by Kostlivy by assuming an abnormal dilatation of the primitive lymph spaces. This would not account for the presence of the lymph nodules and abundance of striated muscle fibers. There must be a pronounced displacement of embryonic tissue. Dowd suggests their origin from Wolffian bodies. This might be true in certain situations, but hardly in all regions in which these cysts are sometimes found.

An attempt to account for them as retention cysts is not possible because of the structure of their walls. Winiwarter was the first to advance this theory. Killian reported a large cyst which he explained by the assumption of an obstruction of the thoracic duct. Bramann believed that the cysterni chyle was the organ obstructed. Kostlivy reported a case which he believed could be accounted for on the assumption of the dilatation of a chyloferous duct. He was unable to demonstrate any obstruction. Tilger's case in which lymphectasis was observed about a healed gastric ulcer is the only case reported in which a possible cause of obstruction could actually be demonstrated. Hlava assumes an obstruction of the afferent lymphatic ducts. This manner of formation may be excluded, according to Prutz and Monnier, by the fact that the abundant anastomosis of the lymph channels would permit

escape through some other channel. Besides Dowd noted that cysts do not develop after obliteration of the thoracic duct. According to my own experiments all that can be accomplished by ligation of the thoracic duct is the production of a peritoneal exudate. It is worthy of note, however, that in elephantatic processes following the total removal of groups of lymph glands areas may be found in which the round cell groups resemble very closely those seen in the walls of the lymph cysts in question. It seems possible therefore that simple stagnation may account for some of the more complicated pictures above alluded to.

The cyst contents of these tumors vary considerably with the structure of cyst wall. The contents may be clear, milky, "chyliferous," bloody, or a mixture of these. The bloody variety is easily explained by the admixture of blood to some other variety of content. The chylous are most easily accounted for on the assumption that they represent chylous ducts. This could only be assumed after it is proved that a duct once functioning becomes obliterated. Most likely this term must be used in a generic sense only as indicating a cyst containing a milky fluid.

A number of cases have been reported as blood cysts which were obviously traumatic or spontaneous hematomata, for instance, one by Brentano in which the cyst followed directly a trauma to the abdomen. Secondary hemorrhage into a preexisting cyst is reported by Wells in which a tumor of the ascending colon known to have existed for 30 years suddenly enlarged. Blood clots were found at operation.

They spring from the central layer of the mesentery but, like myomata of the uterus, later shift their position. Prutz and Monnier have represented these possibilities diagrammatically. It is easy to understand by reference to these how by extension they may compress the gut. Though usually ovoid in form, they are not always so, as is indicated by a case reported by Vautrin. In this case a polycystic mass spread over the entire mesentery like a multilocular air cushion. A combination of the two types may occur in that globular tumors made up of many separate cysts may exist side by side, as I observed in one case.

No predilection as to site seems to exist. Metting in 18 cases



found 11 situated on the right side, while Haln found 8 on the left side to three on the right.

**Pathology.**—The cyst contents are usually clear fluid. In some instances it is mucinous, as in cases reported by Tillaux and Werth. In color these may be greenish, as in the case reported by Tillaux, or yellow as in Thornton's case. Any of these forms may be complicated by hemorrhagic exudates, particularly in those cases in which blood stasis occurs from pressure of the tumor.

The walls are composed of fibrous tissue interlaced with elastic fibers. Muscle fibers have been described. The cysts are lined with flat or cuboidal epithelium. The latter form is likely to occur in tissues hardened in alcohol and evidently is influenced by the contraction of the surrounding tissue. In some places there is apparent stratification. It is difficult to distinguish between the lining cells and round or endothelioid cells in the connective tissue about them. In one of the specimens I examined the picture of endothelioma arose in this way.

**Symptoms.**—In both my cases the tumor was discovered in the course of operations for other conditions. They seemed to be of no clinical significance. In most of the cases recorded the presence of a tumor was the first thing that the patient noted. Sometimes pain precedes the discovery of the tumor. Frenzel notes that it is the sudden enlargement that leads to acute pain. This pain is the more severe the nearer the cysts lie to the root of the mesentery, according to Carter. Küster reported a case in which symptoms of intestinal obstruction were the first manifestation. When hemorrhage takes place into the cysts reactive phenomena attended by sudden severe pain may follow and an inflammatory condition be simulated, as in a case reported by Babler. Many, on the other hand, run their course quite painlessly. This is particularly true of the pedunculated variety. Those situated interstitially, particularly when near the intestinal border, may produce an intestinal stenosis by direct compression. Sudden enlargement of the cyst may lead to acute obstruction. Twisting of the ileum about the cyst led to this disaster in Fertig's case, and also in Briddon's.

Pressure on other organs may produce symptoms. When the

cyst is situated in the pelvis the bladder may be irritated. Gusserow ascribed dysmenorrhea to a cyst in one case.

**Diagnosis.**—The chief physical sign is the presence of a globular tumor of great mobility. Usually it can be made out to be cystic. This mobility distinguishes it from retroperitoneal cysts. Ovarian tumors with long pedicles may simulate them. Usually mesenteric cysts possess a greater mobility toward the diaphragm than ovarian cysts and the manipulation of them does not impart movement to the uterus. Cysts situated near the root of the mesentery may simu-



Fig. 216.—Lymph cyst of the ileocecal region. This mass was accidentally discovered during an operation for gallstones.

late a retroperitoneal cyst or an encysted peritonitis very closely. Pain with cyst, particularly if evidence of obstruction is present, speaks for mesenteric cyst.

**Prognosis.**—The collected statistics of results following treatment are wholly unreliable. This is in part due to the fact that many of the recorded cases were treated before a dependable operative technique was developed. Here it is the operator rather than the disease that requires prognostication.

Arékion records 24 cases of cures to 2 deaths treated by marsupialization. Speckert records 8 recoveries in 22 cases treated by the same method, and Braquehay records 93 per cent recoveries by resection. Bégouin recorded 68 per cent recoveries and Friend noted 12 recoveries and 6 deaths in cases treated by resection. Gildermeister in 51 cases treated by various methods reports that 43 were cured.

**Treatment.**—If an inexperienced operator should unexpectedly encounter a complicated mesenteric cyst it may be well for him to remember that simple puncture has been advocated. Bégouin was a most enthusiastic advocate of this plan even in experienced hands.

With modern technic marsupialization or extirpation is the method of election. The cyst wall may be stitched to the incision either with or without freeing the wall from its environment as much as possible. Terrillon recommends the former procedure. Speckert warns against incision of the cyst if fever is present for fear of the spreading of the infection. In such instances the cyst should be stitched into the incision and opened after adhesions have formed. If the wall is too thin to admit of this, a tampon may be placed over and about the summit of the cyst until adhesions have taken place. The advantage of this plan of treatment lies in its simplicity. The danger of injury to the gut and of hemorrhage is avoided.

As objection to this method of treatment Prutz and Monnier have mentioned the following: continued secretion from the cyst, thus reducing the patient; the gut is fixed thereby inviting to torsion, malignant tissue may be allowed to remain; existent compression of the gut may not be fully relieved.

Resection of the complete cyst is the ideal method of treatment. This is easily carried out in small, simple cysts either with or without resection of the gut. Terrillon and Hahn would limit resection to the simple varieties. The chief factor to be determined before deciding on this method of treatment is the relation of the cyst wall to the larger vessels. Not only must the immediate results be calculated on, but possible late disturbance of the circulation threatening the integrity of the gut it supplies.

### **Endotheliomata**

There has never been a tumor described that could be said to have sprung from the peritoneal endothelium. The endotheliomata of the pleura most likely spring from the lymphatic endothelium. There are many curious tumors observed in the peritoneum, the source of which can not be demonstrated. Some of these present large syncytial masses and are accepted as arising from the uterus, notwithstanding that the history does not bear out the assumption of relationship. Such things make an accurate historical account of tumors of this tissue impossible. The earlier literature contains isolated case reports of primary cancer of the mesentery, but these accounts are not definite enough to exclude a primary focus elsewhere. The diagnosis of a primary colloidal tumor taxes our credulity, and from the nature of things makes us feel positive that a primary tumor elsewhere was overlooked. Sprangenthal, Corsswell and Hodgkin were the first to present apparently authentic cases of primary tumors of the peritoneum. Most of the cases reported in the earlier literature are not based on careful post-mortem studies and some even are without any autopsy at all. Thus Lebert reports 10 cases observed in his own practice. Glocker made a careful study of the literature and could collect but 16 cases.

It is perhaps assuming an unnecessary added burden by attempting to classify these tumors under this head, for no class of tumors in recent years has been the subject of so much discussion as endotheliomata. A great difference of opinion has prevailed regarding these tumors in general. Its greatest height is reached in the discussion of the primary tumors of serous membranes, because here the problem is much complicated by the divided opinion as to the nature of the cells covering the serous surfaces. The histologic status of the covering cells has been discussed in the section on histology and need not be repeated here. The opinion was there expressed that so long at least as embryologists are divided on the point of origin of these cells it is useless to shift the anatomic and pathologic classification to meet these changing opinions. In pathologic as well as histologic discussions it seems best to treat these cells and the tumors they produce in an objec-

tive way. How they look and what they do and not their origin is what concerns us. It is but a name at most that is the subject of the controversy. The final solution of the problem in tumor genesis is better served by a study of facts than by the aligning of observations behind one or the other of several hypotheses.

What efforts have been made to straddle the difficulty by means of a comprehensive nomenclature is well seen by reference to the literature. Schultz, as the title of his paper indicates (*Das Endothelcarcinom*), was uncertain as to the classification. He states, however, that he uses the term "krebs" in a strictly clinical sense. This author believed that the tumor cells were derived from the endothelium of the lymph vessels. Bostrom, Glockner and Teixeira de Mattos adhere to this view. Kolaczek applied a term well descriptive of some types, namely, plexiform angiosarcoma. Boehme gets around the problem by using two terms tandem, as indicated by the title of his paper (*Primäres Sarkocarcinoma der Pleura*). Hokmokl comes a little nearer committing himself. The title of these papers and the descriptions they contain indicate that they had to do with perivascular lymph endotheliomata. Therefore, while they are tumors of the peritoneum, strictly speaking they are not tumors derived from the peritoneal endothelium.

Another group of tumors were aligned with reactive processes by a number of writers. Perls and Birsch-Hirschfeld called them lymphangitis proliferans. Neelsen gave a more oncological ring to his term, namely, lymphangitis carcinomatosa, as did Schottelius before him. I feel a deep personal sympathy for both these terms. Glockner evidently had a more pronounced specimen when he conceived the ponderous term *Endothelioma lymphangiomatosum carcinomatodes*. He recognized, it may be said, that this term was applicable only to the less fully developed forms. Volkmann in a very complete study expressed the opinion that the tumors were derived from endothelial connective tissue cells. Hanseemann supplied terms enough for all contingencies, in carcinoma endotheliale, sarcoma endotheliale and even carcinoma sarcomatodes endotheliale. Such terms as these no doubt would be pleasing to dermatologists and obfuscated pathologists, but they are irksome to surgeons who must think clearly.

We may start out with an understanding that these tumors are

derived from flat cells within the serosa. Since the lymph vessels have been proved to be, like the blood vessels, closed channels, the reference to endothelial lined connective tissue spaces may be omitted. No one has demonstrated the origin of the tumors from the surface cells. Zeigler states that they do, but he does not attempt to prove his statement, as do Jürgens and Napp. Even though these may finally be proved to have some genetic relationship with epiblastic cells, this fact need not concern us here. The mere fact that these tumors produce astonishingly variegated cell structures should stimulate us to determine the fundamental type rather than to cover each variation with a ponderous designation.

**Pathology.**—The multitudinous nomenclature above noted gives a clue to the variability of their structure. Schatteliuss noted that the fundamental difference between endotheliomata and carcinomata lies in the fact that the former springs from many areas simultaneously, while the latter begins in a circumscribed point and extends to the surrounding tissues, or to the regional lymph glands. Gephard reiterated this observation. Nevertheless Desplats and Harris believe that carcinoma may begin at many points at the same time.

The majority of tumors of serous surfaces which have been described were located on the pleura. Since there is an identity of the anatomic structure between the pleura and the peritoneum, the anatomic appearance of tumors of both regions may be expected to be similar. This is the more justifiable since both pleura and peritoneum are sometimes simultaneously affected by tumors of the same histologic appearance.

In the simple form in both these regions there is but a piling up of the endothelium lining the lymph vessels. In the pleura the lymph plexus may be accurately outlined by the proliferating endotheliomata. These sometimes seem to bear some relation to acute irritative processes. I saw such a case in a patient dead from a septic abortion in which a generalized endothelial proliferation was present, most marked in the peritoneum of the broad ligaments. This same condition may be observed in outlying districts of more marked neoplastic formations. This type gives the impression of a generalized hyperplasia of the endothelium excited by some irritant in the lymphatic circulation. This impression is

heightened by the fact that the transitory lymphangitis following peripheral infections of the extremities shows a similar piling up. The dividing line between the reactive and neoplastic does not seem to be a sharp one.

In the pleura this hyperplasia is sufficient to outline the lymphatic network of the pleura as perfectly as can be done by injecting methylene blue. Such lymph channels present a thickened endothelium, usually two to four cell layers deep.

The type yet more pronounced presents small nodules to which the term "miliary" has been applied with a measure of justification. In these the nodular areas present a more pronounced piling up of cells. The markedly tumorous type presents tumor masses of varying size. These masses tend to displace the surrounding tissue, rather than to invade it, thus growing true to the usual type of endotheliomata.

In the latter type the cell strands may cease to show the hollowed gland-like columns due to the proliferation of the endothelium, but instead present solid cordons of cells. The picture of carcinoma is then complete. This differs from carcinoma growing into a lymph vessel, as is sometimes seen in carcinoma of the breast, by the fact that in the latter the endothelium remains about the cell columns. In such instances the cell columns lie closely together with but sparse connective tissue between them. Such specimens present a close duplicate of the ordinary carcinoma simplex.

In another variety the cells do not form nests, but extend tandem between connective tissue bundles. In such cases the cells usually vary much in size but are often very large containing an abundant protoplasm with large spheroid nuclei in which lies a deeply staining nucleolus. These cells resemble very much groggy endothelioid cells as one sees sometimes in chronic reactive processes, notably in hyperplastic inflammations of the cecum and in woody phlegmon of the neck. As before mentioned the amount of protoplasm may be so great as to resemble syncytiums. The same type of cell is observed in other conditions, however, notably in the metastasis of an ovarian tumor.

According to Braude the elastic fibers about the tumor nodules

are much increased. I have found this true likewise in chronic reactive processes.

**Symptoms.**—The most constant manifestation is ascites, as Spencer-Wells has pointed out, and next in importance is pain. The ascites is particularly significant, according to Spencer-Wells, if accompanied by rapid loss in weight. This ascites does not differ from that present in secondary carcinomata but does differ from hepatic or cardiac ascites in that it contains 4.5 to 6 per cent albumin while only .3 per cent is found in the latter conditions.

Thomayer sought to differentiate between this and ordinary ascites by noting that instead of the symmetrical dullness produced by the ascites it extends up more pronouncedly on the left side. This is supposed to be due to the thickened omentum. This is probably true since the same phenomenon is observed in ascites due to tuberculosis in which the omentum is regularly thickened. Due to the same factor is the lessened cardiac dullness. According to Gordon, however, the diminished cardiac dullness is due to the loss of elasticity of the lung.

Pain is not as constant in this condition as in secondary carcinoma, since pain is not marked unless neighboring nerve plexuses are invaded. In cases in which intense pain is associated with diffuse infiltration it is often difficult to determine if there is a primary epithelial tumor or not. This is even more emphatically true of obstructive symptoms.

**Treatment.**—No treatment is of avail.

### **Enterocystomata**

Under this caption are included cysts which are derived from some abnormality of development of the gut tract. They are to be distinguished from mesenteric cysts, which develop independent of the gut tract. Moynihan confuses the problem by classifying mesenteric cysts according to their origin. Lewis and Thyng on the other hand have sought to establish an anatomic basis for the enterogenetic type. They are distinguished from diverticuli by being without connection with the gut tract. Their structure may differ so much from that of the gut tract that according to Nioso, their nature is often overlooked even when examined microscop-



ically. In that event they may be mistaken for true mesenteric cysts.

**Location.**—Owing to the extent through which the omphalomesenteric duct travels and the various changes it undergoes, a variable site is easy to understand. In the abdominal portion but four cases have been reported. The most frequent site corresponds to the location of Meckel's diverticulum. There are nineteen of these. Only three are reported on other portions of the ileum. Five are reported at the ileocecal valve and seven near it.

**Pathogenesis.**—The least likely theory is that of Hedinger who assumes that they are derived from the esophagus because they are often lined with cubical epithelium. More plausible is the theory that these cysts are derived from the fetal gut tract by diverticulation of the gut tract from the fetal epithelium. Gfeller and Sanger supported this view. In some instances the close relation to the gut epithelium substantiates this view. The most probable source is from the ductus omphalomesentericus and Meckel's diverticulum. The chief argument for this theory is the seat of the tumor as above noted—either in the abdominal wall or at or near the usual site of the diverticulum. Raesfeld was the first to suggest this possibility. Hendee, Rimbach, and Colmers support this view.

The occurrence of multiple cysts, Prutz and Monnier point out, could hardly be explained by this view. Kostlivy's view that these are due to diverticula is probably correct. Roth reports a case interesting in this connection. In a newborn child he found a spherical enterocystoma which still communicated with the small intestine. The upper portion was shut off from the lower narrow portion by numerous constrictions. True diverticula with their own separate walls occur, as I have observed. Further proof of their association with the gut wall is demonstrated by a specimen I once studied in which small cysts located in the mesentery were associated with multiple adenomata of the gut wall. That there is a close relation to Meckel's diverticulum may be indicated by the fact that these associated adenomata bore a very close resemblance to the gland tissue often found in the blind end of Meckel's diverticulum. All this means that these structures represent early

stages of gut epithelium, sometimes derived from the gut wall, and sometimes from Meckel's diverticulum.

The fact that these cysts differ in structure within rather wide limits indicates that they represent anlagen derived from the gut tract at varying stages of development. In the case reported by Roth the type of epithelium in the part that communicated with the gut showed a more mature epithelium than that portion which had been constricted off. Perhaps the degree of deviation from the normal gut epithelium furnishes some index as to the time when the anlage was separated from the gut tract.

This variation is so great that in some instances the classification of an enteromesenteric cyst may remain doubtful. In some instances the anatomic relation of the cyst is as valuable a guide in the identification of these cysts as the structure of their walls. This is certainly true when the structure of the wall has been much changed by inflammatory processes.

The explanation of the occurrence of ciliated cells is found in the cases reported by v. Wyss and Dittrich. In the early gut tract ciliated cells have been described by a number of observers and it is the suppositious persistence of these that accounts for the existence of ciliated cells in the cysts.

**Pathology.**—Enterogenetic cysts have been confused with cysts of other regions so that the literature is needlessly confused. Lewis and Thyng have done much to clarify the problem and Miller has recently presented an excellent summary. In the typical cases it appears the relationship to the gut is at once apparent. The form of the lining epithelium Miller suggests is dependent on the intracystic pressure. In the typical cases the villi are retained, while in the other extreme even the distinctly columnar arrangement of the cells is lost. In some even a stratified cuboidal epithelium is found. The wall of the cyst may retain a double wall of muscle fibers arranged at right angles to each other. In the less typical cases irregular bundles of muscle fibers may be all that remain to indicate the origin of the wall. In the latter instance they may resemble the mesenteric cyst of extraenteric origin.

**Symptomatology.**—The symptoms produced by these tumors have to do exclusively with interference with the fecal circulation. In none has a clinical diagnosis been made. In one case the tumor

was so large that normal delivery was made impossible. Of the reported cases fifteen were accidentally discovered at autopsy.

**Prognosis.**—In the thirty-five reported cases twelve died. Eleven cases were operated, of these four died. Two of these were operated as movable tumors of the abdomen, notably those of Rimbach and of Morton. One case was discovered accidentally while removing an inflamed appendix from a hernial sac. (Hendee.)

**Treatment.**—Resection of the gut together with the cyst seems to be the logical treatment as was done in one case by Sudler (Fig. 217). Should inflammation or intestinal obstruction exist, drainage of the cyst with or without enterostomy might be indicated.

### **Lipoma of the Mesentery**

Mass accumulations of fat are not unusual in the mesenterics in fat people. Their unusual enlargement has been reported as lipomata. The mesosigmoid particularly is sometimes the site of great masses of fat. The mesenteric attachments to the small gut are sometimes so greatly distended with fat that the gut is partly surrounded and at first sight may appear as a tumor.

True lipomata of the mesentery are very rare, if indeed a true case is recorded. Alsberg's case grew between the layers of the gastrocolic omentum and became attached to the colon so firmly that the gut wall was lacerated when removal was attempted. A portion of the tumor extended retroperitoneally and was attached in the region of the kidney. This portion was calcareous. This fact strongly suggests that this was the oldest portion of the tumor and therefore represented the site of origin. This case therefore should be grouped with the retroperitoneal lipomata. Waldeyer reports an autopsy performed on a body in which a huge lipomyxoma developed in the root of the mesentery. Since this huge tumor englobed the right kidney, though declared to be mesenteric in origin, it seems to belong where Proust and Treves place it, namely, with the retroperitoneal tumors. These authors abstract thirty-six cases under the heading of mesenteric lipomata. Many of these certainly had their origin in the retroperitoneal connective tissue as will be shown in the consideration of retroperitoneal lipomata. It may be argued that some of these tumors began in

the mesentery and later extended to the retroperitoneal tissue. This view is based on the erroneous view that the mesentery is composed of two layers, and hence a tumor growing between them may separate them.

More authentic are the following cases: Roux reports a case of fibroma and lipoma evidently in the mesosigmoid, and also one in the mesentery of the small intestine about which a volvulus had occurred. Heurtaux reports a myxolipoma in the mesentery of the small intestines. Lennander reports a 15 kg. tumor arising



Fig. 217.—Mesenteric cyst. (Museum University of Kansas Medical School.) Note the flattened gut passing over the summit of the tumor.

from the transverse mesocolon. Madelung reports a case in which the mesentery of the small intestine was involved to such an extent that resection of the gut was necessary.

The few cases of true intraperitoneal lipomata reported presented but little data that would make a clinical picture of distinction. At operation the exclusion of retroperitoneal tumors is about all that is required.

In several of the cases extirpation was successfully carried out.

Before such an act is decided upon, its relation to the retroperitoneal tissue should be determined.

### Secondary Peritoneal Cysts

In areas subject to irritation peritoneal cysts are occasionally observed. These are seen most frequently on the broad ligaments and on the fundus of the uterus after acute salpingitis, and in hernial sacs.

In the former situation their genesis may be followed with exactness. A fibrinous exudate forms over a granular mass, this mass becoming absorbed, leaving a space formerly occupied by the mass. Usually this process leaves but sheets of newly formed peritoneum and cysts form only when the area is enclosed on all sides.

In hernial sacs the opportunities for observation present themselves less frequently and their exact genesis is open to doubt. Tiny cysts within hernial sacs are frequently observed, but larger ones are not so common. They appear as sacculated hydroceles within the wall of the hernial sac or project from its surface. I have seen them within the sacs of hydroceles of the cord associated with hernias. Cantas reports a case in which a cyst the size of a pigeon's egg and one the size of a cherry were found in the inguinal hernial sac in a boy of sixteen who had had the hernia since early youth. This author noted that the portion of the sac wall from which these cysts sprang was in a state of active inflammation.

I have twice noted cysts associated with inguinal hernias in the female. Each was the size of an orange and lay just within the inguinal canal. They were alike in that both lay just within the inguinal ring and had attachment along the course of the round ligament. Opening into them caused me fright, fearing lest I had inadvertently opened into the urinary bladder. Only careful search convinced me that I had not made such a blunder. Resection of the cyst wall apparently resulted in a cure in each case.

Tirket's case seems to have been of a different nature. His patient was a male aged fifty-seven in whom a cyst was noted in the hernial sac. During the following year the patient's abdomen gradually enlarged. After incision the omentum mesentery and

parietal peritoneum were found to be studded with innumerable small cysts. A portion of the omentum was removed for examination and they were found to contain a clear fluid containing mucin and a little albumin. The inner layer of the wall bore cilia.

### Embryonal Cysts

Inclusional tumors in the abdomen are relatively rare. Taruffi reports 71 cases. Braquehayé collected 104 cases, Moynihan collected 113, and Dowd brought the number up to 136. Many more have been reported in the literature, but a number of these are of doubtful diagnosis, while others obviously were derived from the ovary.

Lexer divides these tumors into three groups: (1) True dermoids, (2) those with undoubted fetal inclusions, and (3) true teratoid tumors.

**True Dermoids.**—This group consists of simple epidermoidal sacs usually placed retroperitoneal. I have observed one in the urachus, this being an argument for the correctness of the view that holds that these cysts are rests from the original body cleft. The arguments applied to the explanation of the origin of mediastinal dermoids may be applied here. In that situation tumors located both above and below the sternum would indicate the origin from the anlage of the cutaneous surface. The analogue between the mediastinal and mesenteric situations is very apparent. Augagneur believes that they develop from the ectodermal rests of Wolffian ducts.

Following Lexer it will avoid confusion to consider tentatively the intraabdominal dermoids in four groups, those intraperitoneal, developing from inclusional defects of the abdominal wall, which therefore are intraperitoneal.

About a dozen cases representing the first group have been reported. These are reviewed briefly in Lexer's paper. The most of these represent very incomplete reports, many of them being mentioned incidentally only in autopsy reports.

The second group contains yet fewer representatives. These are located in the retroperitoneal space. Herrera's patient was a male, aged eighteen, in whom the tumor extended from the diaphragm to the pelvis. This was successfully extirpated. In König's pa-

tient, a female aged forty, the tumor lay beneath the liver and was operated on in the belief that it was an echinococcic cyst. The cyst was marsupialized and the patient was dismissed with a discharging fistula.

A third group was first suggested by de Quervain's case in which tumors extended from the retrorectal space to as far as the level of the umbilicus. I once observed a patient in whom three dermoids occupied the retrorectal space, the upper being situated as high as the sacral promontory. These were discovered by following an ordinary sacral dermoid into the hollow of the sacrum. Martini reports a case in which atresia of the anus and urethra was present and the tumor filled out the entire pelvis and communicated with both the bladder and rectum.

The fourth group is less definitely defined, since they exist along with similar tumors of the ovary. Whether these arise from rupture of the ovarian cysts and the dissemination of some of their parts, or whether various parts of the body suffered simultaneous embryonal displacement is not certain. Heinecke's case was a woman of 31 in whom, besides a dermoid of the ovary, one the size of an apple lay under the diaphragm. The only evidence of a genetic relationship of the subdiaphragmatic tumor to that of the ovary lay in the presence of small cysts extending from the diaphragm to the broad ligament. Perhaps more convincing is Kolaczek's case in which a large ovarian dermoid was complicated by numerous yellow nodules, from one of which a tuft of hair grew into the free peritoneal cavity. Fränkel's case was a similar one in which numerous small cysts were distributed along the route to the mesocolon, diaphragm and liver. Lexer reports a case with the tumor situated in the region of the cecum and of a more complicated structure, in that it contained several teeth.

**Fetal Inclusions.**—Only those tumors containing a fetal organ are included in this group. They are situated usually either in the transverse mesocolon or in the epiploic bursa. These anomalies are accounted for, it will be remembered, by Ahlfeld, by supposing the simultaneous development of two embryos. Marchand on the contrary believes that they are developed from a misplaced group of cells, the misplacement taking place in the very earliest period. About a dozen cases belonging to this group have been reported.

One of the earliest and most complete was reported by Young, in which a well formed fetus lay in the mesocolon of a newborn child. This case is particularly interesting from the fact that the inclusion obtained its nutriment directly from the aorta of the host. Pigni reports a case in which a cyst was similarly located and contained a fetus representing about the fourth month. Ahlfeld also quotes cases reported by Fattori in which the cysts were located in the transverse colon. It is interesting to note that in all these instances the anlage must have been situated at a definite point. In one case an interesting slight variation is noted. Reiter and Steinger cite a case in which the sac lay below the stomach, and extended downward and to the left. Obviously the anlage was situated somewhat differently than in the cases above quoted. The point of origin obviously must be in that portion which later becomes the transverse mesocolon. Evidently slight displacements of this anlage by the development of the spleen and pancreas determine its subsequent topographic relations. In this way the inclusions may be covered by the developing cecum, as in Rizzoli's case. Situated somewhat higher in the mesogastrium the anlage would follow that portion of the mesogastrium which goes to form the epiploic bursa. In such instances the tumor may be associated with the duodenum, as in Highmor's case, or it may lie in the bursa itself, as in Bernhuber's case. If the anlage lay at the point of dorsal intersection of the mesogastrium its subsequent growth might lift the peritoneum above it and thus appear retroperitoneal, as in Buhl's and also Phillip's case.

The fact of the definite location of these inclusions would seem to speak for Marchand's rather than Ahlfeld's theory of origin. No attempt has been made to determine the reason these inclusions reach a certain size and then cease to grow. Perhaps a study of their circulation would give some clue.

**Teratoid Mixed Tumors.**—These tumors are distinctly atypical both as to their location and structure. They represent at times all three of the embryonal layers, sometimes only one or two of them, and sometimes they are wholly atypical in structure. It has been established by Roux and others that displaced embryonal tissue is capable of continued growth in its new location. These tumors are usually retroperitoneal. Their origin may be hypoth-



ecated from any of the sources discussed for fetal inclusions. Gross and Baraban report one situated in the abdominal wall, near the anterior superior spine on the right side. In Dickinson's case the tumor occupied a similar position in the right side. Marchand's case lay in the space between the kidney and aorta of a thirty-three-year-old man. Fillaux's case lay in the iliac fossa of a twenty-two-year-old woman, and extended to the renal region. Pilliet's case lay over the vertebral column, reaching from the promontory to the epigastrium. Montgomery's case, a girl of twelve, presented a tumor which lay about the cecum and extended lateral to it nearly as high as the liver. Lexer's patient, a girl of eleven years of age, presented a tumor below the liver.

**Symptoms.**—Tumor was the single feature which excited the attention of these patients. It was only exploration or postmortem that was able to give a solution. The surgeon will do well if he recognizes the character of his tumor after he has it in hand.

**Treatment.**—In the case of the cysts marsupialization may be done if the removal of the cyst wall is too difficult. In the teratomata removal alone is permissible. Obviously in many of the cases recorded any sort of treatment would have been without avail.

### **Tumors of the Retroperitoneal Space**

Tumors beginning in the retroperitoneal tissue are comparatively rare so far as we may judge from the number of cases recorded in the literature. Whenever one is confronted with a condition the diagnosis of which is exceedingly uncertain and its treatment is fraught with difficulty or disaster, it is more than probable that the number of cases reported bears a very uncertain relation to the number of cases that actually are observed. Few men take any great pleasure in reporting a case in which the diagnosis was wrong and the treatment a failure. When one was wrong and becomes right there may be some sense of satisfaction in proclaiming it. But when one has made a blunder and never finds out just what happened it makes a poor text for a paper. One learns more of this by quiet listening in the smoking room than from the forum or library.

From the foregoing it seems worth while to take stock of the

available knowledge on these tumors purely from its practical aspect. Quite aside from this they have a fundamental oncologic interest in excess of their practical importance. Unfortunately most of the cases recorded are lacking in detail to so great a degree that the information conveyed aids but little in enlightening the theoretic problems involved. It is only when the practical problems are enlivened by their theoretic aspects that complete details become available. The two fundamental factors of subsequent history and complete histologic examination are lacking in many of the cases recorded.

Few clinicians understand what a careful histologic examination implies. To illustrate this point I will mention an incident that occurred during a lecture of the late Dr. Fenger. He was demonstrating a large retroperitoneal sarcoma. He had removed blocks for microscopic sections from no less than twelve separate places. After describing slides from all these areas with a detail with which only he was capable, he pointed his finger straight at me and asked, "Now we know what this tumor is made up of?" I had been impressed with the unusual thoroughness and I unhesitatingly gave him my assurance that we were now possessed of full knowledge. "No," he fairly yelled, "we only know what is in those twelve places." It has taken me twenty years to learn the significance of those remarks.

The series of tumors going out from this region because of the names attached are taken to be widely separated in their clinical behavior. Lipoma is the acme of innocence in tumor disposition while sarcoma sounds the knell of despair. In this situation there are often histological gradations. Lipomatous tissue is often intermingled with myxoid, and the latter is always closely related to sarcoma. It is this tendency to admixture that removes them from the usual class of histoid tumors. The size of the fatty tumors often causes them to directly menace the patient. Because of these factors the clinical aspect of these tumors must be considered from a different viewpoint than is usually accorded tumors bearing such designations.

Were it not for harmony in utilizing the recorded cases it would seem best to discard the accepted nomenclature altogether and designate the entire group of tumors occurring in the retroperitoneal

space as mixed tumors. This not only represents the state of our knowledge regarding them but also the structure of most of the tumors. In order to conform as much as possible with the literature the terms lipoma and sarcoma will be retained. As occasion arises an attempt will be made to point out their relation to the teratoid tumors.

**Retroperitoneal Lipomata.**—Under this caption are included all those tumors which are predominatingly lipid in structure. For the majority of the tumors recorded this is clearly a misnomer, for a number will be found in this list which were recurrent and a number in which sarcomatous areas were recognized. Myxoid or “edematous” areas were almost the rule.

**Etiology.**—In five of the recorded cases, among which may be mentioned Neumann’s and Lauwers, the tumor began in early childhood. None are recorded in the second decade of life. This might suggest a congenital anlage, since this corresponds to the age in teratoid tumors of this region and for mixed tumors of the kidney. The fact that lipomata are most frequently observed at the midperiod of life does not argue against an embryonal relationship since adrenal and other tumors of this region present a similar age relationship. The proneness of lipomata to begin in the pararenal region, the frequent site of teratomata, would lend additional weight to this view.

In a few instances trauma has preceded the discovery of the tumor. Tilmann records a case where trauma preceded the development, or at least the discovery of the tumor. Vander Veer records one in which disturbance began soon after conscious injury from lifting, and Homans one following severe bodily exertion. In the majority of instances the insidious onset precludes the possibility of establishing a definite period of time for its beginning. Because of this both age incidence and relation to trauma may be much obscured.

In harmony with relative frequency in the sexes in other regions of the body retroperitoneal lipomas are observed more frequently in women than in men. According to the collected statistics of v. Vegesack in 97 cases in which sex is given there were 72 females to 25 males. This corresponds very well to Grosch’s, who found in 665 superficial lipomas 441 were in females.

**Pathogenesis.**—As the name implies these tumors develop from the retroperitoneal tissue. Obviously they attain their origin from tissue which has been disturbed in its development for a considerable area. This is indicated not only because of their structure and diffuse development but because many of them bear relation to the intramesenteric connective tissue, as is manifest by their tendency to insinuate themselves into the intraabdominal spaces. As already noted tumors developing from the retroperitoneal connective tissue tend to grow into the fossæ in this region and into the retrocecal and retrocolonic connective tissue and in some of the mesenteries. The gut may be displaced far beyond its normal confines, and by this displacement a primary mesenteric origin may be erroneously assumed.

Péan was the first to emphasize the paravertebral connective tissue as the most frequent site of origin. Göbel described the area bounded by the iliac and psoas muscles, which extends up to the lower pole of the kidney, as the most frequent site. As unusual sites the retrorectal space may be noted, as in the cases of Chiari and Neupert. Vander Veer records a case which had a bulging in the back and at autopsy the tumor was found to be attached to the kidney capsule. Johnston reported a case in which the tumor seemed to have sprung from the broad ligament. The vast majority, however, seem to find their chief attachment in the paravertebral space about the height of the kidney. The site of origin has likewise a genetic interest.

The site of the origin can be pretty certainly determined by noting the site of the chief blood supply. König, on the basis of a case in which a tumor weighing 22 pounds was removed and in which at autopsy a secondary isolated tumor was found in the region of the kidney, was led to formulate the hypothesis that there is a primary diffuse anlage extending from the space of Retzius through Douglas space along the ureter to the kidney region. Lexer made a similar observation. In many of the case reports it is noted that secondary tumors extend for some distance from the main tumor. Homans noted that these lipomata were prone to be formed from many lobulations. In one of his cases there was a secondary lobe situated some distance from the main tumor, connected only by a fibrous band. In nine of the reported cases more or less extensive

lobulation was noted. In Schiller's case the number of accessory lobulations suggests a relation to the peripheral symmetrical fibrolipomata. This is further suggested by Roux's case in which one of the accessory tumors was fibroid in character. It would not seem that we should expect anything different since lipomata in other situations likewise are prone to accessory lobulations. This is particularly noteworthy in the case of the lipomata in the abductor muscles of the thigh. Here the accessory lobules often extend long distances between the muscle planes or even into the muscle substance itself. These tumors are particularly interesting in this connection, for, judging from their tendency to secondary metaplasia, they stand very close to the retroperitoneal lipomata. Neupert records a case of a characteristic lipoma of the thigh which extended through the obturator foramen, establishing a continuity with a tumor in the pelvis. In one case reported, lipoma of the thigh was followed by a like tumor in the retroperitoneal space of the lumbar region. Since that followed within six months of the removal of the primary tumor a relation is possible.

The more rapid growth of these tumors as compared with lipomata developing on the surface can be explained by their close relationship to the sarcomata. Ebner suggests that the lesser resistance the tumors encounter in the retroperitoneal space might account for their rapid growth. This might be assumed in the beginning, but when they become large they no doubt encounter more resistance from the abdominal wall than from the loose skin unreinforced by muscle. Their more rapid growth can more consistently be ascribed to their proclivity to myxoid and sarcomatous degeneration. Their riotous rate of growth would almost warrant the revival of the ancient term "malignant lipoma." According to Vegesack 42 of the recorded cases showed myxoid change. In six cases sarcomatous admixture has been recorded. Whether the occurrence of sarcoma takes place in the connective tissue, as suggested by Vöckler, or from metaplasia of the myxoid tissue is difficult to determine. From my observations of analogous conditions in the thigh I should lean to the latter possibility.

All these facts point to the probability that these associated conditions receive their anlage from the beginning and that retroperitoneal lipomata represent a very embryonal type of tissue, and

that rapid growth, myxoid and sarcomatous admixture are but manifestations of a primary impulse. The simultaneous occurrence of these various tissues seems expressive of a compound tumor rather than a degeneration of a lipoma. The inherent close relation to malignancy is even more apparent when the retroperitoneal sarcomas are considered.

A clear recognition of this fundamental problem is desirable. The idea of lipoma makes surgeons bold. Experience has proved that disaster attends action on this concept. If the close relation to teratoid tumors were recognized, surgeons would hesitate more before proceeding to radical operation in the more advanced cases and thus save disappointment or disaster.

**Pathology.**—Lipoid tissue with more or less intermixture of fibrous tissue constitutes the usual structure. In this they differ in no wise from lipomata of other situations. Their chief claim to distinction lies in their disposition to be associated with myxoid tissue. In half of the cases collected by Vegesack there was a combination with some other tissue. It may be noted in passing that in many of the cases where its presence was not noted an altogether insufficient microscopic examination was made and in many of the cases the diagnosis was made on clinical grounds entirely. It seems difficult to determine in a given instance whether the association with their tissues shall be regarded as a degeneration or whether both classes of tissue exist from the beginning and grow side by side. The latter is probably the case. They also differ from the usual type of lipomata by the disposition to form long arms which insinuate themselves into neighboring spaces. In this tendency they differ from superficial lipomata. A similar tendency is noted in the lipomata developing in the abductor group of muscles of the thigh. For reasons already stated it is quite possible that the relation of these to the retroperitoneal tumors is closer genetically than those of the subcutaneous tissue.

The myxoid tissue complicating these tumors is likely to become sarcomatous. In fact in many of the rapidly growing lipomata a hyphenated relationship to the more malignant tumors may almost be assumed to exist as a matter of course. Neupert reported a case that lends weight to this view. Other cases have been recorded by Vegesack, Waldeyer, Gerster and Vöckler. Since the

malignant areas may be small, prolonged search may be required to find them. This is particularly true of the myxoid areas. I once used a tumor of this sort as a supply of material for the purpose of demonstrating the structure of a pure myxoma to a class of students. Finally an area was found distinctly sarcomatous in character. Inquiry disclosed the fact that the tumor contained sarcomatous areas, the patient having died of a recurrence.

Cyst formation sometimes noted is probably due to liquefaction of myxoid areas, though in a few cases an association with cavernous lymphangiomata may be assumed. Borst reports a case in which he believed the cysts were due to retention of lymph. Degenerative changes have no doubt accounted for some of the cyst formations. Von Vöckler reports a case in which a smooth-walled cyst resulted from myxoid degeneration. Secondary changes of other characters are sometimes observed. Most notable is the deposit of calcareous material, as in Madelung's case. At least half a dozen of the recorded cases have shown such calcareous deposits. Vestberg (quoted by Ebner) found highly organized bone.

**Symptoms.**—In all the reported cases the onset was insidious. It was only after the tumor had attained a considerable size that it was recognized. Preceding the discovery of the tumor there were usually symptoms of an indefinite character. Most frequently a sense of fullness, often associated with progressive weakness, was complained of. Symptoms even more indefinite, as sleeplessness, eruptions, and cardiac irregularity were occasionally noted. Later more distinct pressure symptoms appear; obstinate constipation, disturbance of respiration, and urinary disturbance, all due to pressure, represent the more definite symptoms. Vesical tenesmus has been reported by a number of authors, notably Roux, Büttner, Gardner, and Adami. These symptoms are due, according to Vegesack, to a retention hydronephrosis.

More directly suggestive are the obstructive symptoms on the part of the blood vessels. Edema of the legs, scrotum, and vulva on the affected side are significant. Even more so is the dilatation of the venous plexus over the lower abdomen. Ascites is mentioned by many, among whom may be mentioned Madelung and Térillon.

When the possibility of the existence of a tumor becomes apparent ascites and the lesions that produce it are simulated. The soft-

ness of these tumors makes early recognition difficult, particularly if in addition fluid is known to exist or associated lesions which are known to be capable of producing ascites.

**Diagnosis.**—Once the existence of a tumor is recognized the question of differentiation arises. Chagrin should not overwhelm the surgeon, for only Terrillon and Schiller (cited by Heinricius) were able to make the correct diagnosis before operation. The general topographic relation to the large bowel is the most important diagnostic point. The gut is carried on the summit of the tumor and usually displaced toward the median line. When this relationship is demonstrated many tumors whose presence may be suggested may be excluded, notably tumors of the liver, spleen, ovaries, uterus, omentum, etc. The lateral location of the tumor tends to exclude uterine and ovarian tumors. When bulging backward, as in Vander Veer's case, extraabdominal tumors may be suggested. When a retroperitoneal location is demonstrated, there remains only the differentiation from other retroperitoneal tumors, notably its fellow group, the retroperitoneal sarcomata. From these it differs in its more diffuse growth, softer consistency, and less definite tumor formation.

From kidney tumors, the absence of any urinary signs, less pronounced bulging in the renal angle, and softer consistency may give the right clue.

Lipomata are so soft that extensive studies relative to their mobility are not possible. Garkische suggests the use of the cystoscope to exclude any renal affection. A definite notion of the capacity of the opposite kidney might be of comfort when in the midst of an operation where one kidney was surrounded by the tumor.

In days gone by when exploratory incision was less safe than now, exploratory aspirations were done—always, of course, with negative results. Because of the inability to locate certainly the hollow viscera, this practice is to be condemned.

**Prognosis.**—Without treatment the increasing size of the tumor tends to destroy the life of the patient, usually within the period of several years from the time of the initial symptoms and a period of months from the time of the discovery of the tumor.

The result of operation is dependent on the topography and size of the tumor and the ability of the surgeon to properly plan his



procedure. So far as the environment goes, aside from the vessels, the neighboring organs may suffer injury. The gut and kidneys are most likely to be injured. Gussenbauer and Chanazan were obliged to remove the kidney because of the close association of this organ with the tumor. Several other operators inadvertently removed or injured the kidney. The gut may be injured directly as in Madelung's case, or the mesenteric vessels may be injured and the nutrition of the gut endangered, as in the case of Alsberg and Roux. Büttner believes that death in one of Tillaux's cases was due to the compression of sympathetic ganglia in the depth of the wound.

According to Vegesack the operative mortality approaches 38 per cent. What is the ultimate fate of those who recover from the operation is in most instances quite unknown. In a number of cases it is specifically stated that recurrence took place with the subsequent death of the patient, either from the tumor itself or from repeated attempts at relief by operation.

**Treatment.**—Without a specific knowledge of retroperitoneal lipomata it is quite natural that the surgeon should approach the removal of such a tumor of whatever size with a great degree of confidence. The recognition of the close relationship of these tumors to the teratoid tumors of this region greatly reduces this rosy view and clinical experience substantiates it.

The smaller the tumor, the greater the prospects for easy removal. The multiple lobulations, some of which may extend to an inaccessible situation or in close proximity to great vessels, makes any operation fraught with uncertainty. Operators regularly report that the operation offered greater difficulties than were anticipated. Homans particularly emphasizes this point. In his cases extra lobulations made complete removal impossible. The difficulties this surgeon encountered are so graphically set forth that one can fairly hear his pantings as he labored valiantly with the huge mass. In Madelung's case also, complete removal was not possible because of the extensive interdigitations with the neighboring structures. In many instances enucleations progressed satisfactorily until the depth was reached when vessels of unanticipated size were encountered. In one case the vena cava was injured in the effort to separate the tumor from it, and one case

is described by Meier in which the aorta and vena cava were inadvertently included in a ligature in the frantic attempt to arrest the hemorrhage. In Neumann's case the blood supply to the colon was destroyed to such a degree that the resection of the gut was necessary.

So far as the technic goes, most operators have selected the transperitoneal route. Ligation of vessels as they are encountered and tamponade of the remaining cavity to serve in the dual capacity of drainage and hemostasis was the usual procedure practiced. Lexer alone was able to close without drainage.

### **Retroperitoneal Sarcoma**

Under this caption are included only those growths derived from the connective tissue and fascia of the retroperitoneal tissues. This excludes tumors growing out from retroperitoneal organs, including the lymph glands. It also excludes those from embryonic teratoid anlagen, since those tumors belong to the group previously discussed. Their uniform structure, to be discussed below, makes it possible to recognize a very well-defined group. Their close relation to the lipomata has been considered.

The first clear description of retroperitoneal sarcomata was written by Lobstein. Not only did he correctly recognize their topographic relations, but also gave a good gross description of them, but he confused them with lymphadenomata and tuberculous affections of the lymph glands. Virchow separated out the true retroperitoneal sarcomata and defined their origin more precisely as from the fascias. There is still a tendency by some to confuse these tumors with those arising from lymph tissue. This error is not justified, for the retroperitoneal sarcomata are in topography, mode of growth, and histology wholly different from the lymphatic tumors.

Keresztszeghy and Steele have collected and discussed the literature pertaining to these tumors.

**Etiology.**—In considering the pathogenesis it is necessary to remember the close topographic relation to teratoid tumors of this region. The frequency with which myxomatous tissue is found in the sarcomata is additional evidence that there is a close relationship to embryonal rests.

The various types predominating at different ages is further evidence of relationship to congenital tumors. In early life, before ten years of age, a very cellular type predominates. Unfortunately in a number of reported cases there seems to have been no distinction between primary sarcoma and the more common teratoid tumors well known to be predominant in this period of life. Likely those tumors reported as retroperitoneal sarcomata in children all belong to the teratoid group. The most frequent occurrence of the typical retroperitoneal sarcomata is between thirty and fifty years. After sixty they are rarities.

Males seem to be slightly more frequently affected than females. The difference is too slight, however, to make it likely that sex is other than a coincident factor.

**Pathogenesis.**—The origin of these tumors is confined to the fascia about the spinal column, particularly at the height of the renal arteries. What has been said relative to the topography of the lipomata of this region may be repeated here, as might be inferred from their close structural relationship. Adami expressed the opinion that all sarcomata are ingrafted on lipomata.

**Pathology.**—These tumors are usually spheroidal with numerous prolongations and lobulations. They are usually soft, sometimes semifluctuating, but may be quite firm if connective tissue predominates. Usually the smaller ones are the most dense. On section they are pinkish white and glistening. Areas of myxomatous degeneration are often seen sometimes in more or less advanced states of liquefaction. Not infrequently complete liquefaction with cyst formation takes place. Sometimes hemorrhage takes place in these softened areas and there result then blood-filled cysts or hemorrhagic infiltration of solid areas.

The typical structure of retroperitoneal sarcomata is identical with the predominant type in the ovary. They are uniform spindle-celled tumors or mixed-celled tumors with loosely arranged connective-tissue bundles. The nuclei are usually of many shapes and sizes, often showing deeply staining clumps as though regressive processes were present. When more active growth takes place, as often happens in an attempt at complete or incomplete removal, the cells are much more numerous proportionately and assume the picture of a round-celled or mixed-celled type.

In the recorded cases many unusual cell types are reported. From some of the reports it is evident that mixed tumors have been included. It is possible that in some situations unstriated muscle fibers may be included, as in the case of McGraw and Steele, but if this tissue is present in abundance, the possibility of the existence of a mixed tumor must be considered.

**Symptoms.**—The onset as in the case of the lipomata is always insidious, there being a gradual onset of pressure symptoms expressed either as pain or edema of dependent parts, particularly the legs or scrotum. Usually investigation instigated by the presence of one or other of these phenomena results in the discovery of the tumor. Pain in the region of the distribution of the nerves may be present. The lumbar region or down the legs are the dominant sites. Sometimes unexplained areas are involved. Ellis reported a case in which in addition to edema of the scrotum the left lung and left side of the face were involved. Steele explains the pain by pressure on the sympathetic system. In this connection it is well to remember that Elliott and Virchow reported cases in which thrombosis of the femoral vein complicated the condition.

Pressure may, besides causing pain and edema, interfere with the function of neighboring organs. The gut tract naturally receives the brunt of such offense. Osler reported a case in which there was polyuria without sugar.

**Diagnosis.**—Retroperitoneal sarcomata must first be diagnosed as a retroperitoneal tumor. This having been established, differentiation from other retroperitoneal masses may be considered. The chief point in determining the retroperitoneal character of any tumor is the determination of its relation to the colon. The tumor arising behind the peritoneum either carries the colon on its summit, particularly if, as is usually the case, the tumor grows into the connective tissue behind the colon and carries the gut on its summit or displaces it to one side or the other. The transverse colon usually describes an arch along its lower border. The position of the colon may be located by inflation, or better still by means of barium and the x-ray.

The stomach is apt to suffer the same displacement as the transverse colon, but is more likely to be elevated than depressed, or pushed to the left, or to the left and downwards.

Retroperitoneal tumors present less mobility than intraperitoneal tumors and move less with the respiratory excursion of the diaphragm.

After all the data have been considered, the exploration by operation is the final deciding point. Fortunate is the observer who is able readily to determine this point once the abdomen is opened.

Once the diagnosis of retroperitoneal tumor is made, retroperitoneal sarcomata must be differentiated from other tumors occurring in this region. Tumors proper to retroperitoneal organs and the teratoid must be excluded.

Tumors springing from the kidney when small and occupying their place in the loin may be distinguished by bimanual palpation with a high degree of certainty even in the absence of urinary findings. The ability to cause the tumor to present to the examining finger at the lower border of the twelfth rib is particularly characteristic. Larger tumors occupying a considerable portion of the lateral half of the abdomen can not be so readily distinguished. The general fixity of the tumor and the displacement of the colon by the tumor may signify its location but its nature may be open to doubt. Metastatic tumors may resemble both renal and retroperitoneal sarcomata. I once observed a striking illustration of this difficulty in diagnosis. A man of twenty-three presented a tumor extending from well under the costal margin above, full to the spinal column medially, and below it rode in the fossæ of the false pelvis. It was irregularly oblong, the lateral border representing the arch of a smaller circle than its median border. The surface was marked by moderate undulations. The region near the upper part was firm but slightly elastic. The lower portion was softer but not fluctuating. One was unable to rock it independently of the skeleton, but it evidently was attached to the fascial structure of the paravertebral region. It presented less mobility than even the large renal tumors usually do. Its extension well into the iliac fossa and its apparent fixation there made it particularly resemble a retroperitoneal sarcoma. Its huge size also indicated the same thing. Besides there were no urinary signs, neither was there any history of there having been any. Notwithstanding the fact that the diagnosis of hypernephroma was made by exploratory incision by another surgeon, the diagnosis of retro-

peritoneal sarcoma was made, but operation obviously was not possible. At autopsy many areas of extensive hemorrhagic infiltration mingled with cellular areas were encountered. Further search discovered the primary nodule in the left testicle. The patient had not mentioned the presence of this tumor and none of the examiners discovered it.

The location of adrenal tumors suggests the differentiation. When an adrenal tumor first appears it presents itself at the costal border of the eighth rib, according to Israel. Sarcomata usually present lower down and are usually attended by fewer symptoms in proportion to their size. There is apt to be paresthesia of the lumbar plexus, often a rise of temperature and in some instances a bronzing of the skin. Israel warns against mistaking compression of the common duct by some other kind of tumor with attendant jaundice for the discoloration due to adrenal disturbance.

Tumors of the pancreas bear much the same relation to the colon as sarcomata. Because of the location of the pancreas above the transverse colon, its tumors tend to bulge above it. The colon is most likely, therefore, to lie along its lower border. Harris found this to be the case in 95 per cent of 34 cases of pancreatic cysts observed. Sarcomata usually lying lower tend to carry the colon on their upper border. This is not true, of course, when sarcomata arise above the pancreas. The high location may bring even the stomach below them. Cysts, the commonest tumors of the pancreas simulating sarcomata, are often accompanied by rapid emaciation, which is not the case in sarcoma. The exact median location is less common in sarcoma than in pancreatic cysts. Solid tumors of the pancreas are usually carcinoma. The dense nodosities together with the general symptoms accompanying usually make the diagnosis easy.

Teratoid tumors of the retroperitoneal tissue are usually observed in children. They are round tumors, dense, or dense elastic, and usually present just below the costal margin. When these tumors appear in later life they are more dense.

**Prognosis.**—Retroperitoneal sarcomata are always fatal. Freedom for several years is sometimes obtained, but recurrence follows.

**Treatment.**—When globular, removal is not attended by unusual difficulties since, being expansile in growth, they tend to press the

large vessels aside. When they present interdigitations which extend among the vessels removal is impossible. Recurrent retro-peritoneal sarcomata are always inoperable.

### **Tumors of the Omentum**

The tumors of the omentum parallel those of the mesentery, as might be expected, but the peculiarities of the function and topography of the omentum give its new growths a certain degree of individuality.

The vast majority of tumors of the omentum are solid, and consist of malignant deposits derived from some other source, but many tumors have their beginning in the omentum. These tumors are necessarily limited to the development of histologically normal tissues, or those arising from the limitless growth of these tissues. To these must be added congenital anomalies involving this structure itself or some organ morphologically more or less closely related. These may be considered in order.

**Lipomata.**—Collections of fat within the omentum are not exceptional. These may assume the aspect of tumors, but so long as they maintain dimensions proportionate to the general adiposity of the individual, they can not be classed as tumors. Huge lipomatous masses which can not be dignified by the name of tumor, may be observed in obese persons. For instance, I once operated on a very obese woman with a huge abdomen. The abdominal enlargement had developed in a period of 18 months without any considerable increase in the general adiposity. An indefinite mass could be felt in the pelvis which was resilient, if not fluctuating. Since dyspnea was becoming progressively worse without evidence of cardiac decompensation, the possibility of the presence of an ovarian cyst was accepted. If that were the case relief by operation seemed a possibility. Since the patient could not lie down, operation was undertaken under local anesthesia with the patient sitting on the edge of the table, the operator sitting between the spraddled legs like the old time obstetricians seated themselves while aiding a parturient woman. When the abdomen was opened I encountered a huge mass of fat which filled the whole abdomen and was at least 6 inches in thickness. It seemed to be the uniformly enlarged omentum. If such a tumor were discovered in a sparse

individual I might well have regarded it as a tumor. Since such accumulations are often seen in lesser degrees and nearly always diffusely distributed over the entire omentum, these had best be excluded from the present consideration.

Minor local accumulations are often noted. Most notable in this category are the hypertrophied appendices epiploicæ. These not infrequently are as large as walnuts and sometimes as large as moderate-sized oranges. Such conditions also are observed only in obese persons, and therefore can not properly be regarded as tumors. Sometimes these masses are associated with excessive lipomatous accumulations in other parts of the body. I recently noted epiploic tags as large as small oranges in a woman who bore masses as large as a quart cup situated about the shoulder and hips and on the medial side of both knees. The subcutaneous tumors bore all the landmarks of true lipomata and possibly the epiploic hypertrophies deserved such an appellation. At any rate the patient was gratified to learn that the external accumulations were lipomata and not "just fat." Malifert (cited by Ebner) reports a case in which a twelve-pound lipoma sprang from an appendix epiploica of the sigmoid flexure.

Much confusion exists in the literature relative to what should be regarded as mesenteric lipomata. Thus Prutz and Monnier discuss under the heading of lipoma of the mesentery many tumors that are unquestionably retroperitoneal in origin. Lower includes Homan's cases in this category. These were chiefly retroperitoneal. He also includes Mendeth's case as well as J. Cooper Foster's. In some of the reported cases retroperitoneal lipomata in their development carry the colon with them and become attached to the omentum, and in this way in a measure insinuate themselves between its layers but in doing so do not, as pointed out by Munro, thereby repudiate the land of their nativity.

Other lipomatous masses become developed within the omentum as the result of accidents. It is not unusual to find huge masses in the omentum in umbilical hernias. I once removed a mass weighing eight pounds from this location. The same condition may be observed in hernias of other locations. For instance, I once removed a mass of fat the size and shape of a cocoanut from an irreducible scrotal hernia in an old man. It had received attach-



ment to the base of the sac and the omental attachment was no larger than a lead pencil. It was dependent quite as much on its secondary attachment for its nutrition as upon its original omental connection. Partial torsion of the omentum may be followed by more or less massive accumulations in the distal part. These may appear as tumors in the vulgar sense, but they bear none of the earmarks of true neoplastic growths and their consideration may be deferred to their proper section. Edebohls reports a case in which an omental mass was adherent to a degenerated fibroid, and another in which such a mass surrounded a recalcitrant tube and ovary.

A case is reported by Legiardi-Laura, in which a tumor the size of a goose egg lay on the anterior aspect of the great omentum which was connected with the omentum by a thin vascular pedicle. Lower's case presented three lobulations in the lower left termination of the omentum.

When all these facts are considered it becomes clear that greater accuracy in reporting this class of tumors is needed.

**Sarcomata of the Omentum.**—Primary sarcomata of the omentum are less common than in the retroperitoneal tissues. The literature is very much confused. Many of the cases reported as such are distinctly endothelial in character. In general two classes may be distinguished, the diffuse, in which the entire omentum is thickened, and the localized, in which a distinct tumor nodule occupies some part of the great omentum. The former is very closely related to the endothelial tumors, the latter to the mesenteric sarcomata.

The attempt to form a definite picture is made much more difficult by the attempt of authors to escape the difficulty by using a straddle term. Thus Miller employs the term "endothelial sarcoma." Happily here, in spite of the misleading title, a cut and an excellent word picture by D. S. Lamb show the tumor to be an endothelioma.

The diffuse variety forms a thickened mass which covers the entire abdominal contents. Cobb reports a case in which the omentum resembled a bath towel, forming a mass an inch thick, covering the intestinal mass. Matas compares his case to a sponge, and notes that it was friable and presented a trabeculated stroma in

meshes of which was a translucent gelatinous stroma. The mass is made up of nodules varying in size from a pinhead to a hazelnut or larger. It is reddish gray in color, friable for the most part, and necrotic in some places.

I confess an antipathy for this group. The whole picture is contrary to that presented by all kinds of sarcoma in any other part of the body. Sarcomata tend in general to develop in an expansile manner. On the other hand endotheliomata in serous surfaces tend to cover wide areas. Before a diagnosis of sarcoma of the diffuse variety is made it should be subjected to the closest scrutiny. Probably more often than has been suspected, they represent secondary tumors, as Boormann has contended. Probably some of the diffuse examples were nothing more than chronic reactive processes. Those which are friable and show degenerated areas are always open to suspicion. Unfortunately the after history of such cases is generally lacking. Without this he must be a brave man who ventures a positive opinion as to their nature.

The typical sarcomata form rounded bosselated tumors, pinkish gray in color, showing a fibrous network on section. Anders reports a typical case of this type in the following words, "large whitish pink, nonvascular, markedly lobulated and furrowed tumor mass."

**Pathogenesis.**—Little can be said about the factors which antedate the development of these tumors. Tate reports a case in which at primary operation an omental mass extended into a scrotal hernia and enveloped the cord and testicle. The thickened mass extended somewhat into the abdomen. This was diagnosed round-celled sarcoma. This was evidently correct, for in sixteen months the patient presented himself with a large mass in the abdomen. The interesting feature is that this patient had previously had his hernia treated by paraffin injections and at operation large masses of this substance were found about the hernial ring. It is well known that paraffin oil has the property of stimulating cell proliferation and there may have been some connection in this instance.

**Pathology.**—The tumors reported have usually presented spindle cells with rather abundant fibrous tissue or interspersed with myxoid tissue. Woolsey reports a case in which the first tumor

removed was reported by the pathologist as a fibroma. A recurrence was recognized as fibrosarcoma. Round-celled tumors occasionally occur. Second reports a case in which a melanotic tumor in an omentum was attached to a uterine myoma. The presence of melanin is explained by the fact that the patient had had an eye removed four years before, therefore it obviously was a secondary tumor. Capelle reports a case of lymphosarcoma. There was an ulcer in the region of the pylorus. The author thinks this was the primary lesion. This would be unusual since sarcomata in that situation are not prone to ulceration. Goldenstein reports a case in which a cystic sarcoma was discovered in the pelvis four years after a sarcoma of the uterus had been removed. The question here, the author thinks, is whether this tumor was primary or secondary to the uterine tumor. He would be quite safe in assuming that it was secondary. It is always precarious business to diagnose a tumor as primary when a representative of the class has existed elsewhere in the body.

The gross appearance of the nodular type is that of a lobulated mass, light gray or pinkish in color. The blood vessels traverse the constricted region. On section they are semiluent and in areas may be cystic.

The diffuse variety is composed of lobulations usually described as varying from pinhead to grape size or larger. In some instances they have been pedunculated. The tissue is mottled grayish red, sometimes hemorrhagic.

**Symptoms.**—Gradually increasing weakness, nausea, vomiting, loss of flesh and strength are the phenomena that usually antedate the discovery of the tumor. Swelling of the feet is sometimes noted but much less constantly than in retroperitoneal tumors. These tumors differ but little therefore from similar tumors arising elsewhere within the abdomen. Cobb made a clinical diagnosis of malignant disease of the large gut. In a case reported by Cabot the first symptom noted was a hematuria, a phenomenon easily explained by the fact that the tumor had invaded the bladder wall.

The tumor is often obscured by a coexistent ascites. When the tumor is diffuse, its demonstration by palpation is more difficult than when localized and nodular.

The onset may be acute, simulating peritonitis. Capelle reports

a case diagnosed as an acute peritonitis. The acute symptoms in this case may be explained by the fact that a recent hemorrhage into the peritoneal cavity had taken place. Such an error would be invited if in addition to pain a leucocytosis should exist. Cobb records a case in which there were twenty-three thousand leucocytes.

The degree of pain varies. Usually it is vague and indefinite. In a case reported by McLean there was pain severe enough four years before to require morphine for its relief. This case is further remarkable by the fact that throughout the source of the disease severe pain was caused by a sudden twist of the body.

**Diagnosis.**—As intimated, the surgeon will do a creditable act, and incidentally confer a favor to oncologists, if he will make a satisfactory diagnosis after operation with all the aids available in such cases.

**Treatment.**—A few have been followed by relief for a period following operation. Cabot, notwithstanding the involvement of the top of the bladder and a loop of the terminal ileum, necessitating the resection of these, secured relief for his patient for at least three years. Bonamy and Bonamy secured a recovery from operation from a large spindle-celled sarcoma. McLean also secured relief for at least three years following a resection of the transverse colon. Woolsey operated three times and at last accounts his patient was free.

Usually, however, the treatment has consisted in exploratory laparotomy with more or less satisfactory exploration. It is very difficult in such cases to determine the point of departure of the tumor.

Douglas lost his case from secondary gastric hemorrhage.

**Fibromyoma of the Omentum.**—The few tumors that might possibly belong under this caption have stirred up considerable discussion as to their genesis. They must needs come either from a gut wall, then not omental in origin, or they must arise from some muscle source in the omentum. Obviously only the vessel walls could be the source. Klebs advanced the theory that myomata of the uterus arose from the vessel walls of the uterus. Lubarsch assumes a similar source of origin in sarcomata of the stomach. Cohen could find evidence of vascular genetic origin in only three

out of fifteen cases. Anitschkow in the examination of myomata of the cordia found evidence for origin about vessel walls. Orloff failed to find any relation between the vessels and the tumors. My own observations in seedling myomata of the uterus have been in accord with this.

**Sarcoma of the Omental Bursa.**—The tumors described in the literature as being native to the lesser peritoneal cavity form a collection from which no definite picture can be formed. Obviously a tumor growing out from any of the contiguous structures and developing into this region may retain little evidence as to its nativity. This is particularly true of the malignant types. A number have been reported, the origin of which has been laid in this region. Gross and Sencert report a case in which the tumor arising from the lesser omentum spread to the greater. Kaufmann described a tumor, the size of a man's head, in the lesser omentum. Secondary masses resembling hydatidiform moles covered the wall of the stomach and great omentum. Walcker reports a large cystic tumor with two distinct cavities the one of which was infected due to ulceration of the mass into the lumen of the stomach. Miodowski reports a case of a solid spheroid tumor going out from this region. Lohfeldt reports three cases. In the first case there was a tumor the size of a swan's egg well encapsulated. On section it was yellowish, translucent for the most part with darker areas. Fibrous bundles divided it into large lobules. It contained a number of small cysts. The capsule was intimately adherent to the stomach and colon. A small opening in the stomach wall communicated with one of the cysts. Two openings united the lumen of the colon with other cysts. It was formed by round cells intermixed with spindle cells. His second case presented a massive tumor of soft consistency and grayish red color which took in all of the left half of the abdomen. Whitish grape-like tumors hung from the diaphragm. One of these smaller tumors was examined and found to be made up of round cells with large dark nuclei. In some regions a limited number of spindle cells was observed. The third case a tumor the size of two fists was situated behind the colon and stomach. The great curvature was united with a tumor, the interior of which communicated through a perforation the size of a finger. The tumor was composed of spindle cells.

From the foregoing it is apparent that these tumors do not differ from other omental and mesenteric tumors and their interest is purely a topographic one.

**Tumors of the Gastrocolic Omentum.**—New growths in this limited field are not numerous. Clarke reported a case in a woman aged fifty, who had an abdominal tumor of four years' standing. A large tumor occupying the space between the layers of the small omentum was found. It was fibroid in structure. Gould reports



Fig. 218.—Wandering tumor of the abdominal cavity. The lower left picture shows the tumor somewhat reduced in size in its normal state. The upper picture shows the same in section about the natural size. The right picture shows a moderate magnification. The central area is degenerated fat, while the upper border shows the capsule of newly developed fibrous tissue.

a case in a male aged thirty-eight, in whom a tense, firm, roundish tumor extended from the tenth costal cartilage along the left semi-lunar line to the pubis, filled out the right groin to the eleventh rib on the right side. The tumor pushed the stomach and intestines into the pelvis. It was successfully removed. Murphy reports a case in which a hard mass extended from the tip of the sternum to four fingers below the umbilicus and laterally into the right and

left hypochondria. It could be moved both laterally and horizontally. The tumor had caused no symptoms. At operation it was noted that the tumor developed from the posterior portion of the gastrocolic omentum and extended downward on the posterior wall of the stomach, elevating the posterior peritoneal layer. Veins as large as the index finger lay on the posterior surface of the tumor. On section it proved to be a fibroma with areas of myxoid degeneration.

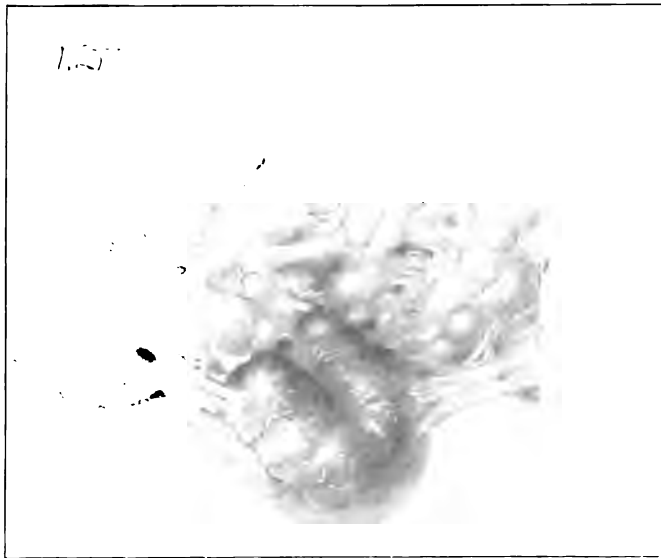


Fig. 219.—Wandering tumor which has secured secondary attachments to the omentum. The structure of this nodule was the same as the preceding but the connecting fibers suggested a recent origin.

### Wandering Tumors of the Peritoneal Cavity

I have seen a number of tumors floating free in the abdominal cavity which resemble very closely similar bodies in the knee joint. It seems likely to me that they are derived from the pinching off of fat tags. When entirely free (Fig. 218) they are covered by a thick fibrous membrane resembling that covering corn pith that has been placed in the abdominal cavity. The interior is made up of fat cells in varying degrees of preservation and sometimes crystals are intermingled. I had one specimen (Fig. 219) in which

the mass was attached to the edge of the omentum by fibrous bands. This attachment obviously was secondary. Many theories have been advanced as to the origin of these bodies. Emmert has recently collected them, together with an abstract of the cases reported, 38 in all.



Fig. 220.—Carcinomatous mass in gastrocolic omentum in a case of carcinoma of the pylorus. Advancement was by direct extension. The great omentum was not involved.

### Secondary Tumors of the Peritoneum

The commonest tumors of the peritoneum are those occurring in conjunction with tumors of other organs. The most common primary seat in such instances is in the stomach, and next most common is the ovaries.



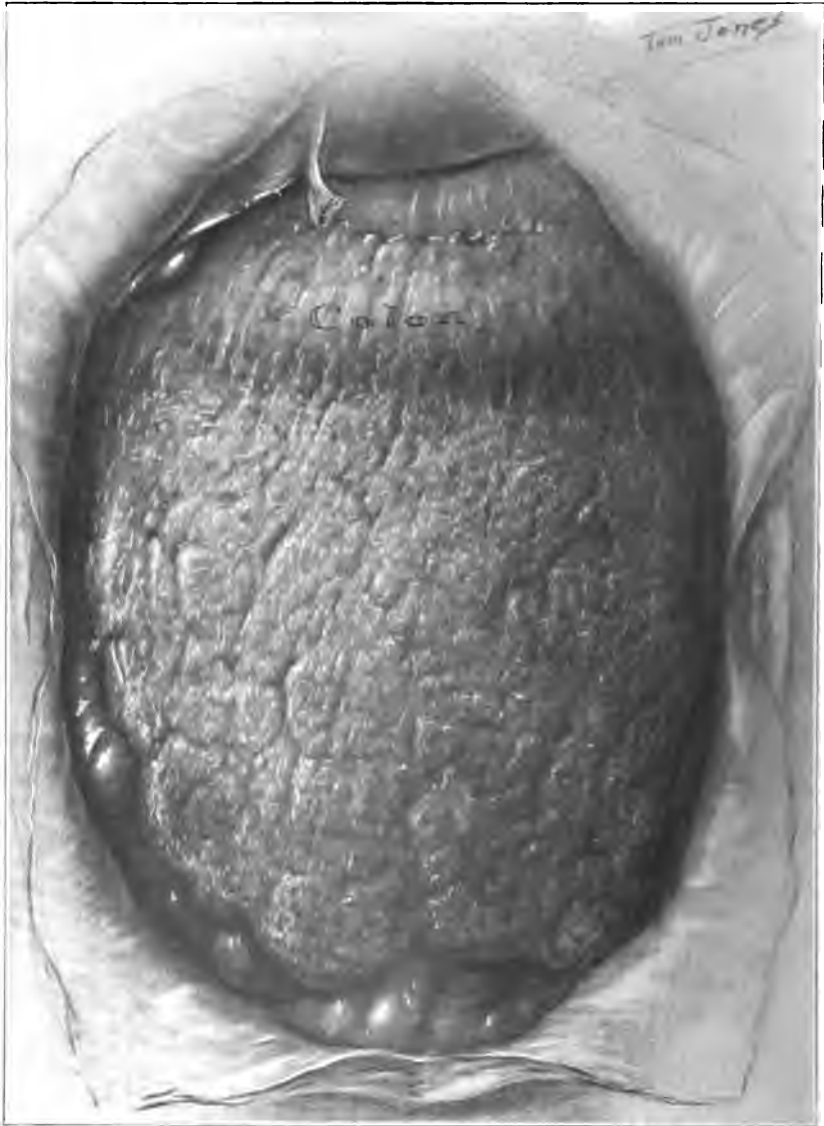


Fig. 221.—In contrast with the preceding the great omentum was converted into a huge mass by secondary invasion of carcinoma secondary to carcinoma of the stomach.

**Pathogenesis.**—In general four varieties may be distinguished according to the manner in which they spread from the primary seat to the secondary deposits in the peritoneum; namely, by direct

extension as in most colloid tumors, by superficial dissemination as in papillary cysts of the ovary by hematogenous dissemination as in diffuse sarcomatosis, and by dissemination with reaction as in pseudomyxoma peritonei.

*By Direct Extension.*—In a great many tumors of the gut tract there is a direct extension to the peritoneum from the affected part (Fig. 220). Few carcinomata of the gut it may be said run their



Fig. 222.—Secondary tumor of the mesentery. A newly developed fibrous capsule covers in the implanted tumor nests.

course without some involvement of the omentum and mesentery and often late in the disease the secondary disease far overshadows the primary growth in pathologic dignity. This extension at first often involves the lymph glands only, but later many nodules may be scattered throughout where no lymph glands normally exist. These secondary implantations may extend by direct growth

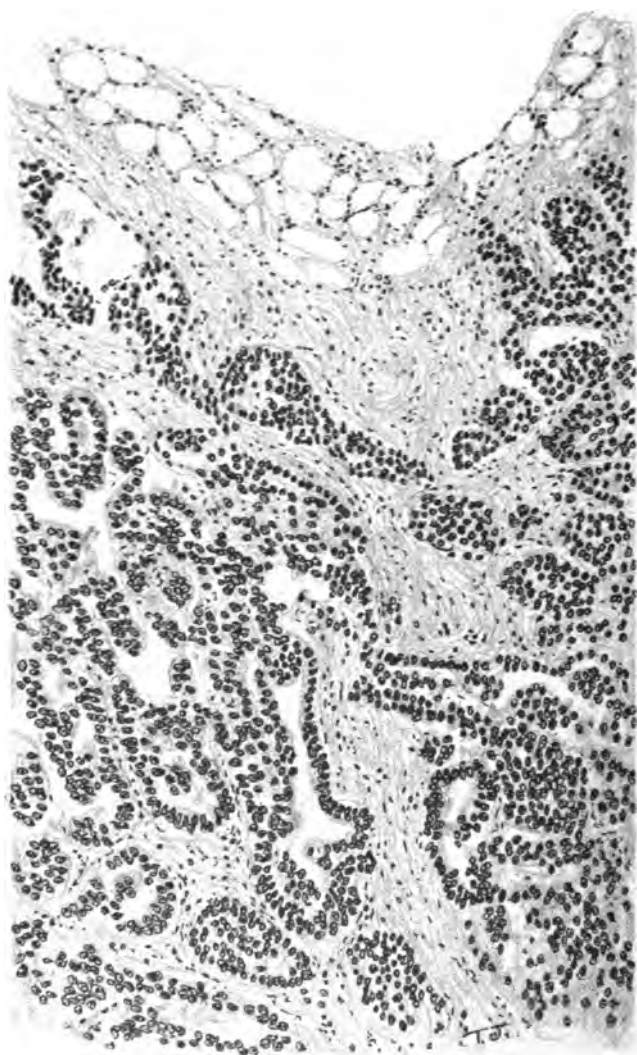


Fig. 223.—Secondary glandular carcinoma of the omentum. The mother type of tumor structure is accurately preserved.

through the connective tissue spaces, or along the lymph and blood vessels. In some instances the entire omentum or mesentery may be changed into a solid mass (Fig. 221). Very often in carcinoma of the stomach large masses are formed in the upper abdominal region which when exposed are found to be tumors in the gastro-



Fig. 224.—Secondary carcinoma of the omentum from a papillary cystoma of the ovary. Cysts containing fluid as well as small papules are formed in tissue otherwise thickened by diffuse carcinomatous infiltration.



Fig. 225.—Secondary carcinoma of the peritoneum of the anterior abdominal wall. In this case the entire surface was covered with nodules causing it to resemble tuberculosis.

colic omentum. This extension may travel somewhat circuitous routes. In a case of hypernephroma I found large masses in the omentum. There was no direct attachment and the possibility of extension through the blood stream must be considered, for in this case the tumor mass extended from the kidney to far up the cava.

*Extension by Dissemination.*—In papillary serous ovarian cysts, often the cyst wall has ruptured allowing the tumor mass to be-

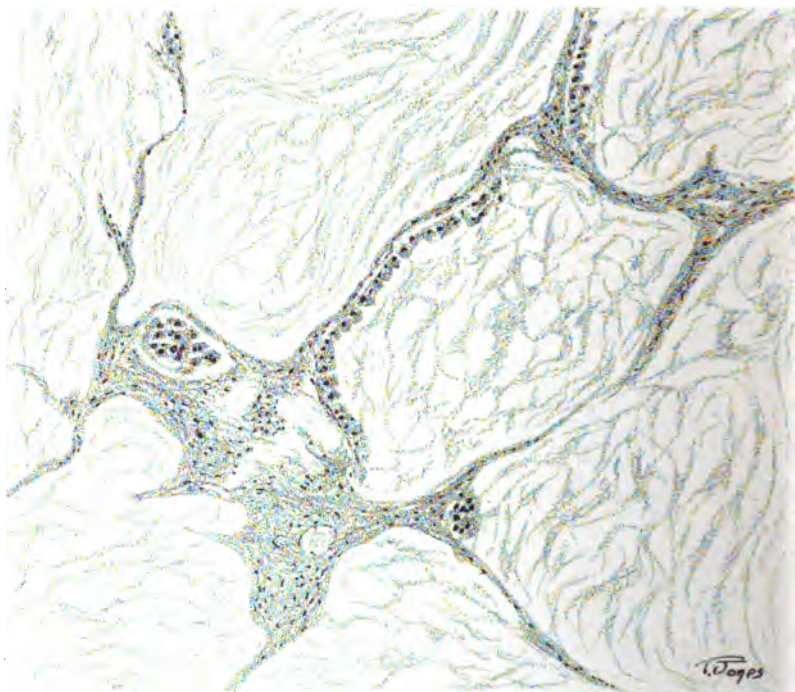


Fig. 226.—Colloid metastasis in the omentum. In most alveoli all cells are degenerated. In others a single layer has been preserved.

come exposed, particles break off and scatter over the peritoneum and there become implanted. These “heal in” just as a foreign body does (Fig. 222). Their presence excites a reaction on the part of the peritoneum which in turn exudes its plastic material and a fibrin network covers the tumor tissue. Growth then continues.

This type is of interest because the mother tissue may be re-

produced in the peritoneum, the glandular (Fig. 223) papillary or even cyst (Fig. 224) formation may be continued in great perfection. Curiously enough these daughters are sometimes dependent on the mother tumors for continued existence, for when the primary tumor is removed the secondary ones may disappear. Unfortunately this takes place only rarely.

*Hematogenous Dissemination.*—In those instances where the en-

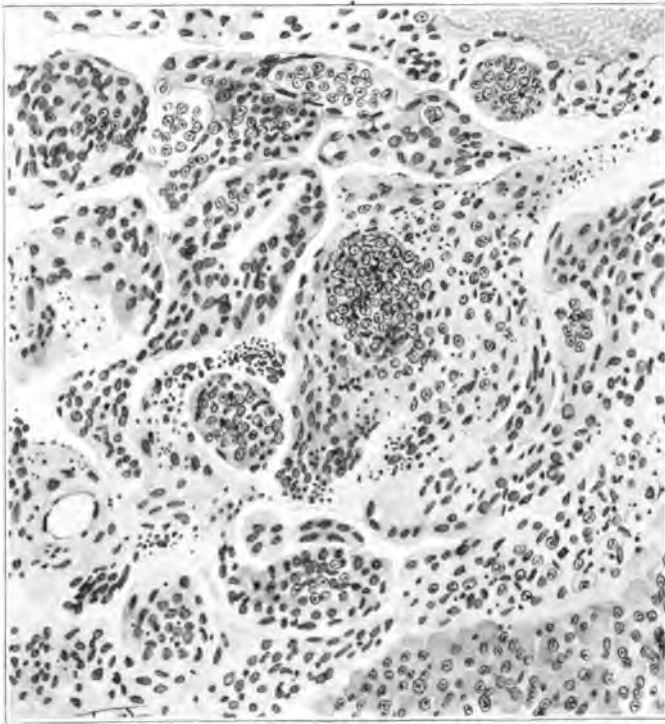


Fig. 227.—Syncytial masses of the omentum secondary to papillary cystoma of the ovary. Some of these nodules closely resemble true syncytioma. There were no such areas in the primary tumor.

tire body becomes studded by secondary tumors, the peritoneum obtains its share (Fig. 225). I recall one case in which a carcinoma became diffusely disseminated. An industrious interne counted more than a thousand in the skin, but when the peritoneum was exposed he gave up his mathematical task in despair. I saw a similar case in which a melanoma of the temple which had been

"cured" by a plaster gave rise to a similar widely distributed tumor formation. Among the curious things in medical literature are those cases of melanotic tumors which are recorded as being primary in the peritoneum. I recall one case in which a melanotic tumor of the eye had been removed three years before, yet the tumors in the peritoneum were declared to be primary.

*Dissemination with Reaction.*—In a small group of tumors the peritoneum reacts against the intrusion of the tumor mass. This condition is called pseudomyxoma peritonei and because of the great interest attached to this condition it is treated as a corollary to this chapter.

**Pathology.**—Secondary tumors of the peritoneum are interesting because of their structure, because they may retain very closely the structure of the mother tumor or may depart astonishingly widely from it. As an example may be mentioned the small epithelial-lined cysts seen secondary to ovarian cystomata. Little less remarkable is the glandular fidelity often seen in tumors of the colloid cells of the gut tract. Here the simple colloid gland cell may be retained with the production of huge masses of colloid material rivaling in mass the pseudomucinous material in pseudomyxoma peritonei. This colloid material may destroy the cells which produced it, at any rate alveoli filled with colloid material but without a trace of a cell, or any debris of them, are found (Fig. 226).

As an example of wide deviation those cases may be cited in which large syncytial masses containing several nuclei are found (Fig. 227). These are called syncytiums by some writers, but where there is no primary source there is no reason for hypothecating them. Personally it does not seem to me unreasonable to assume that these syncytial masses are the product of the endothelial cells due to irritation by tumor cells of other genesis. I have seen several instances of this kind occurring in association with papillary cystomata of the ovary.

In some instances there seems to be no structural relation between the primary tumor and the secondary tumor in the mesentery and omentum and such relation can only be hypothecated on general grounds. For instance in a case of hypernephroma with associated tumors in the mesentery it is only ordinary caution, based on

a knowledge of the variety of multiple tumors of primary origin that causes us to accept such a relationship despite the fact that there is no structural similarity.

**Symptoms.**—When a malignant tumor secures a secondary establishment in the peritoneum it manifests itself either as a mass or by exudation. The appearance of the mass is usually superimposed on the symptoms produced by the primary malignancy. This is not always the case, however, particularly in carcinoma of the stomach. A mass in the gastrocolic omentum may appear before there is any evidence of gastric distress. Exudate may appear as the first symptom, particularly when secondary to papillary tumors of the ovary.

**Diagnosis.**—Secondary tumor masses are dense even in the coloidal type and are usually so typical that their discovery clears up the diagnosis, if any existed before. In the case of a dominating exudation there may be some difficulty even after the peritoneum is exposed. This is true only in the case of miliary dissemination because of the close resemblance of these nodules to tubercles (Fig. 228). If the primary tumor is recognized, the differentiation may be easy, but even the recognition of the primary tumor may not be easy. A localized tuberculosis of the gut may be surrounded by tubercles which closely resembles a carcinoma with local dissemination. I have seen a number of instances in which a laboratory examination was necessary. Age is no criterion for I saw a carcinoma of the transverse colon in a boy aged eighteen with dissemination that resembled a tuberculosis very closely. In some of these cases a degree of care needs to be exercised even in the examination of the section, for in tuberculosis the endothelial elements may so dominate the field that a malignancy may be suspected, and on the other hand a malignant cell nest may be so profusely surrounded by round cells that a very young tubercle may be diagnosticated. The most helpful sign at the operating table is the shape of the nodules. Tubercles are often oblong and may show tiny bosselations while the cancer nodule growing expansively does not have these nodules and may show some tendency to umbilication. Usually, too, in carcinoma there will be some nodules too large to classify as tubercles.

Sometimes fluid obtained at operation or by aspiration may show



large epithelial cells which may aid in the diagnosis of malignancy. The fluid from tuberculosis of the peritoneum shows a predominance of lymphocytes.

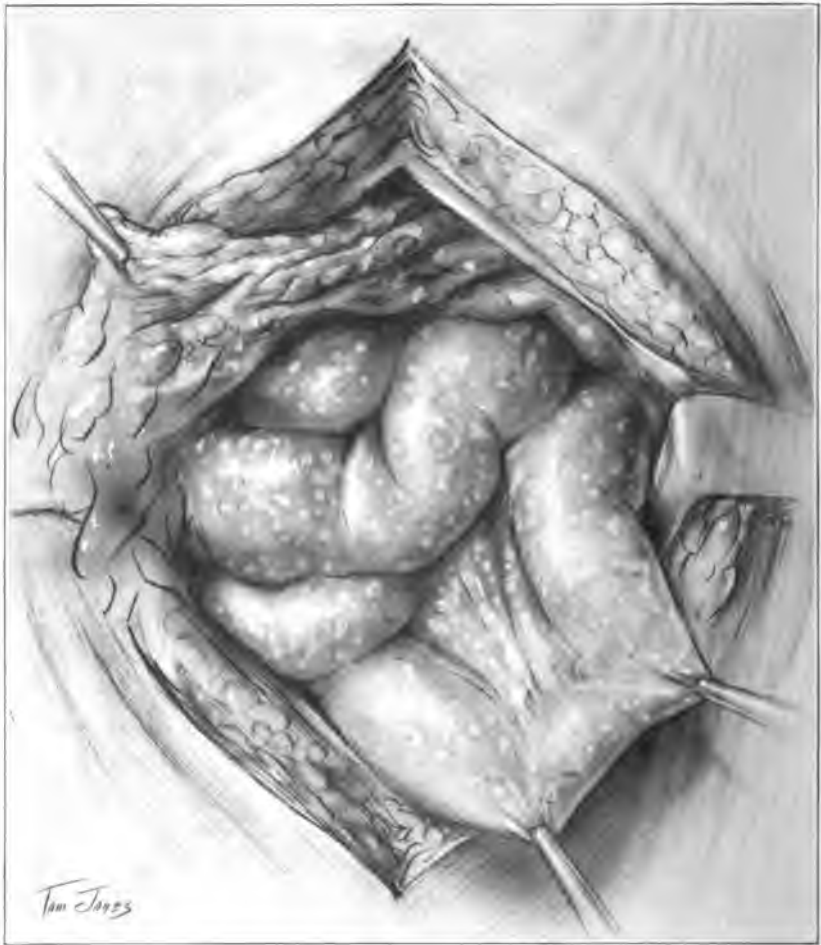


Fig. 228.—Miliary carcinosis of the peritoneum. Some of the nodules show dimpling. There is less tissue reaction than in tuberculosis.

**Prognosis.**—Save in case of carcinomata following papillomata of the ovary, the prognosis is absolutely bad. In the case of these if the primary tumors can be removed and the secondary tumors are young, the removal of the primary tumors is advisable.

Carcinoma of the peritoneum in conjunction with solid tumors of the ovaries does not admit of any treatment. These tumors are often associated with carcinomata of the stomach, and death usually follows within a few weeks if any treatment is undertaken.

**Treatment.**—In addition to the remarks just made, in extensive exudation temporary relief may be obtained by withdrawing some of the fluid.

### **Pseudomyxoma Peritonei**

Under this title is described a condition of the peritoneum in which masses of gelatinous material are diffusely distributed over wide areas of the peritoneum, either as a homogeneous layer or as cyst-like masses. This condition follows bursting of ovarian cysts or cystic appendices, and possibly other tumors. The relation of this material to the peritoneum may be purely a passive one; or it may be responded to by the usual reactive processes, or real secondary tumor proliferation may occur, or a combination of these.

It is more questionable whether the term employed fits all these conditions or any of them. On the contrary, the term should be regarded as purely symbolical and in no wise be regarded as prejudicing the pathogenesis. In the first place a negative attribute furnishes a poor basis for a designation. In the second place the name given the condition is based on an erroneous conception in that the substance under discussion is mucoid and not myxoid. If we were sure that that substance was identical with the product of the cysts the condition arises from, we might employ the term pseudomucinoma peritonei. Until such a time, however, as we shall have a proper term for pseudomucin we may as well employ the conventional term above accepted.

In a number of instances in the older literature cases have been described which set forth more or less clearly the condition now being considered, as for instance, Atlee describes a case that may belong here. Schroeder and Péan each report several probable cases. More clear are the cases reported by Beinlich. It is worthy of note that Salkowski recovered mucin from one of the cases. Menning noted its association with ruptured ovarian cysts and studied the reaction produced on the part of the peritoneum, and

expressed the opinion that this membrane became active in the production of the substance. Menning was the first to present a microscopic study. Though Werth gave no better description than those above noted, he is generally accredited with having first adequately described this disease. His chief distinction lies in having mistaken its pathogenesis and assisted in perpetuating an erroneous appellation. He did emphasize the fact that the existing factor is the bursting of an ovarian cyst with the resulting pouring out of the contents of the cyst, though Virchow had previously expressed the same opinion. His term was regarded as an improvement on that employed by Virchow, namely, "myxomatous degeneration of the peritoneum." Olshausen contributed to the pathologic conception of the disease in that he expressed the opinion that it was a metastatic and not a degenerative process. Pfannenstiel confirmed this opinion, basing his opinion on the study of four cases.

The collective literature may be found in Strassmann's article which presents a collection of 36 cases, and Schumann's who collected 20 more. This last paper presents the best recent collection of literature.

**Pathogenesis.**—The disease under consideration follows the rupture of a pseudomucinous cyst and the escape of its contents into the peritoneal cavity. Whether by its presence this material sets up a reaction on the part of the peritoneum, producing the completed picture of the disease as Virchow (quoted by Beinlich) was the first to advance, or whether cells from the cyst, finding a new nidus in the peritoneal cavity, continue to perform a nefarious function, constitute the two theories of the genesis of this disease. Olshausen was the first to advance the theory that the condition does not represent a simple reaction of the peritoneum to irritation, but that it actually represents a neoplastic process.

It is not easy to select between these two theories. Olshausen's theory unquestionably is correct for a part of the cases, for in some of them true pseudomucinous tumors are scattered over the peritoneum. It is equally true that in others no trace of epithelium can be found and such cells as are found can not be identified with certainty.

When an attempt is made to align the analogous conditions aris-

ing from the appendix the difficulty is heightened or clarified according to the leanings of the observer. The appendiceal type seems incapable of producing the cysts such as follow the rupture of ovarian cysts. Lejars, with much show of reason, argues that the condition is due to a pouring out of the goblet cell secretions into the peritoneal cavity. Goldschwend believes that the cells extruded when the appendix ruptures are dead and incapable of further development.

In some cases columnar epithelium is found in the lining of cysts secondary to pseudomucinous cysts of the ovary. This may occur as well when there is no escape of cyst contents. Baumgarten records a case in which many cysts filled with colloidal material were found in the peritoneum after the removal of a cystoma of the ovary. These cysts were lined with columnar epithelium. This may take place a long time after the removal of the cyst. In Olshausen's case the removal of the original cyst preceded the peritoneal complication by 17 years and Elizabeth Lewis records a case the interval in which was 22 years. I once observed a patient from whom I removed bilateral pseudomucinous cysts in which there was no rupture and no reason evident at the operating table to cause me to be apprehensive of a recurrence. Examination of the walls after the completion of the operation showed malignancy in both tumors. In two years many small pseudomucinous tumors covered the pelvic and abdominal peritoneum. These were clearly metastatic tumors just as one sees them following papillary tumors of the ovary, but they were of course morphologically different. It is obvious that peritoneal metastasis may follow pseudomucinous cysts either with or without rupture. Whether this represents merely a complete development wherein the more typical pseudomyxoma of the peritoneum represents a less fully developed form is open to question. Netzel (quoted by Strassmann) is of the opinion that the ovarian tumors which give rise to pseudomyxoma peritonei differ from the usual pseudomucinous ovarian tumors. He would call them ovarian myxomata.

The evidence of proliferative activity of the cells which escape with the cyst contents is not great but positive. In case of the appendix Goldschwend is of the opinion that the cells which escape with the colloidal masses are dead and incapable of develop-

ment. Experience seems to bear out this assertion, for in none of the reported cases has proliferation of the epithelium been demonstrated. McConnell's case may be an exception. Here granular proliferation of the wall of the appendix at least seems to have been present.

In case of the ruptured cyst contents there are undoubted evidences of proliferation. Fränkel particularly emphasizes this point, though the areas he demonstrated are small and none too plain and he prejudices his case by saying had he searched more thoroughly he undoubtedly would have found other areas. The use of the subjunctive mode always jars in the description of scientific observations. However, Olshausen and Gebhard both attest to the fact that epithelial proliferation takes place.

While observations of these authors may be accepted, there are a number of directions in which the conclusions may by far overreach the minor premise. Because some of the material shows evidence of proliferation the conclusion is not warranted that all do so, nor does the mere presence of cells, even with evidence of active proliferation, warrant the conclusion that the pathologic process is all or even in part due to such activity. Their presence is far too sparse to warrant the assertion that the process is to be regarded as a simple metastatic process. Fränkel even goes so far as to advise the abandonment of the title which implies activity on the part of the peritoneum. In none of the four cases from which I was privileged to examine material could any evidence of columnar celled activity be found. Perhaps I labored under the ban of the subjunctive mode as set forth by Fränkel, but energy and credulity often run parallel. Another source of error is in classing frank metastatic tumors of the peritoneum with the disease under discussion. In this condition there are real cysts which may burst, it must be added, and thus they may simulate pseudomyxoma peritonei. This phase of the question can best be studied in the section dealing with peritoneal metastasis of colloidal tumors in general.

The source of the abundant material is not explained by the small number of cells present in the extruded material. Perhaps Lejars has the correct notion in that he expresses the belief that in the case of the appendix the cells continue active, and produce

material which from time to time is added to that already extruded. This might be assumed in the case of ovarian cysts likewise when the cyst with a patent opening is still present, but in those cases in which the cyst has been removed this view fails. In those vast areas in which fibrinoid septa unite masses of homogeneous material to the peritoneum one must assume that the cells that produced it have degenerated and their carcasses have become blended with the homogeneous substance, or that some of the mass is the product of local tissues. The use of strong solutions of formalin-glycerine applied to the peritoneum may produce a material macroscopically very much like that in pseudomyxoma peritonei. It is true that the more thorough the investigations the fewer cases in which epithelial cells are not found. There are instances in which known carcinoma of the stomach is followed by the production of colloidal masses in the omentum and peritoneum, which on section give very small evidence of cell activity. Ewing reports a case in which the association with the gut tract was discovered only after careful search.

Even in the most carefully investigated cases, however, the number of cells seems ridiculously small compared to the amount of myxoid tissue. Often in large masses of material no cells are found. If cells produced this, their complete disintegration followed. The formation of web-like strands of fibrous tissue without cells and without full development of fibrous tissue resembles very much the changes that take place in the hemorrhagic exudates in myomata. That a considerable reaction on the part of the peritoneum does take place is well attested to by the fact that it led Virchow to believe that the reaction was the source of the material. This phase may more properly be considered in the paragraphs on pathology. It may be mentioned here, however, that we have become so thoroughly imbued with "*omnes cellula e cellula*" that we can scarce think in other terms. When a tumor grows in a tissue it compels the tissue to give up the material necessary for the elaboration of its kind. It seems to me that in this instance the mucinous material coming in contact with a serous membrane compels this membrane to give up a like material. The mucinous material acts in this instance as a ferment in that it perpetuates its influence. In the large colloid myomata we must depend on

some such process. It is possible to find every stage of change from small vascular degenerations to large cysts. Columnar epithelium is never found here. There is no use sticking to the fetish of cell secretion in this case, for we know nothing of the chemical methods in any cell secretion.

That such is possible is in line with the opinion of Eichwold who compared the cement substance between endothelial cells with the mucin of ovarian cysts, and Schaffer regarded this substance as mucin. If we regard the opinion of these authors as correct, a source for the substance in question is present.

In accounting for the presence of the colloidal material in this connection, it is worth noting that in primary colloidal carcinoma of the peritoneum Zeigler (1895) believes that the colloidal material is derived from the endothelium of the blood and lymph vessels. Glockner also believes that it is derived from this source. Miller and Wyss believe mucin found in this condition is a specific fluid, being like joint fluid. The term colloid is morphologically and macroscopically descriptive of several products of cell activity or degeneration that have nothing in common except their gelatinous character. So long as we have no trustworthy knowledge of the chemical composition of substances, the product of endothelial and epithelial activity, we can but hypothecate its origin from its association with one or the other of these types of cells.

If we possessed reliable chemical knowledge of the peritoneal material here considered some aid could be expected. Beinlich reports that Salkowski found mucin in one of his cases. In all the cases in which chemical analysis has been made, all except that reported by Neubaur contained mucin. If these analyses withstand the tests of modern chemistry, the presence of this substance might serve as a guide in comparing this substance with that developed in ovarian cysts. The fact that ovarian cysts contain no mucin, hence called pseudomucinous (Pfannenstiel), some differentiation might be possible. It would be interesting to know also if those cases following rupture of the appendix are of the same composition as those derived from ovarian cysts. McCrae and Coplin report a case that according to them probably had its source from the gall bladder. I have in several instances removed gall bladders

filled with mucin. These showed no change in the epithelial lining, but did show changes in the wall of the gall bladder that would admit the thought of colloidal degeneration. McCrae and Coplin state that in their case the morphologic and chemical reactions of the columnar cells were identical with those of ovarian cysts. The gelatinoid material from the peritoneal cavity was examined by Hawk and pronounced "much like the material known as serosa mucin."

It is worthy of note that the appendix may be involved secondarily to the ovary. Eden reports a case in which two years and four months after operating for pseudomyxoma peritonei going out from the right ovary, a renewal of the disease was found to affect the left ovary, which was healthy at the time of the first operation. More important is the notation that the appendix was distended with colloidal material.

It is worthy of note, also, that the necessary condition for the production of a pseudomucinous appendix is the occlusion of the lumen in the proximal portion of the appendix. Chavannaz records a case in which repeated attacks of appendicitis were followed by a pseudomucinous appendix. This is so thoroughly in accord with gall-bladder occlusions with subsequent distention that further argument is unnecessary. These facts at least point to some relation of reactive processes to the production of the condition under discussion.

The next problem is whether or not those ovarian cysts which give rise to pseudomyxoma peritonei differ in structure from the usual pseudomucinous cysts.

The cysts which antedate the peritoneal condition are universally stated to have friable walls. Rupture is most apt to occur, as Spiegelberg pointed out, in those in which there are many daughter cysts which grow to a size to press on the walls of the mother cyst. It may possibly be worth noting that it is in just these cases that the most active cellular proliferation is present. These, therefore, stand closest to the papillary serous ovarian tumors in which secondary implantation is the regular thing.

In many of the case reports it is impossible to gather from the description of the microscopic specimen what the character of the cells may be which are found in these tumors.



Attempts to solve the problem by experimentation were abortive. Donati introduced some of the material obtained from a case, into the peritoneal cavity of a rabbit. He had previously placed this material in a solution of carbolic acid. The animal died 5 days later. He noted that the mass was surrounded by a "pus celled" membrane and the peritoneum surrounding it was similarly covered. Beyond this the peritoneum was hypertonic. My own experiments in this line gave quite similar results save that the "pus membrane" was seen to be a fibrinous layer in which were many polynuclear cells. The pseudomucin may be regarded, therefore, as an irritant producing a greater degree of reaction than for instance a bit of corn pith.

When all has been said, there is still a lamentable lack of definite knowledge. The outstanding fact is that in some of these cases there is a definite metastasis. In other instances small islets of columnar cells only are found, while in others cells of uncertain genealogy are found. These facts warrant the general opinion that the condition is the product of cell growth derived from the cyst. Those arising from the appendix fit less kindly into this scheme. The instances in which cell proliferation was demonstrated are few and unsatisfactory. Chemical knowledge which would enable a comparison is lacking. That reaction on the part of the connective tissue is marked will be seen in the discussion on pathology. Whether this reaction is merely a protest against the growth, or whether it contributes to the mass of mucoid material which irritated it to reaction, only more definite knowledge can decide.

**Pathology.**—The structure of the tissues produced by this disease presents a number of perplexities all of which are not yet solved. The essential factors to be determined are to what degree the peritoneum suffers changes, and to what extent the cells extruded from the cyst are capable of autogenic activity.

Testimony is in accord on the point that when the abdomen is opened large quantities of colloidal material roll out (Fig. 229). This material is usually widely distributed, being usually insinuated even between the liver and diaphragm. It is usually described as being yellowish, honey-like in color, though it may be, as I have observed, nearly pearly white. If any unusual activity or traumatism has occurred, it may be tinged with blood in certain places,

which may appear as bright red streaks of recent hemorrhage or in diffused chocolate colorations of more ancient extravasations. The colloidal material may be diffusely distributed in homogeneous layers, but usually, at least in some areas, tends to assume globular

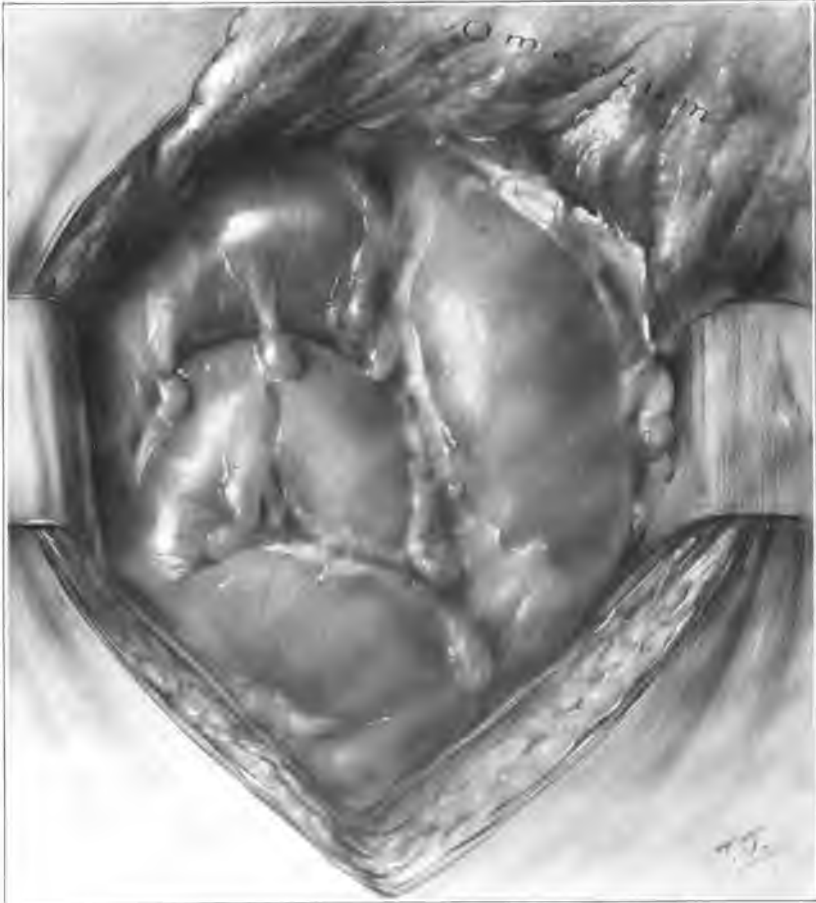


Fig. 229.—Pseudomyxoma of the peritoneum. Large masses were scooped out with the hands before the intestines were exposed.

forms, either in response to physical laws or to developmental processes. These globular masses may be covered with fine vascular pellicle-like membranes. The physical character of this material is well expressed by Virchow when he states that strings of it may

be drawn out with forceps and cut off with the scissors. What he fails to state, however, is that the pieces so cut off tend to assume a globular form. This physical property may best be demonstrated by causing it to flow from the abdomen or from a container, or by forcing some of the material through the clefts between the fingers of the closed hand. Like molasses, when the volume is not too large, a globular form is attempted by the isolated masses. The difficulty of removing this material from the abdominal cavity at autopsy or at operation is due in part to its viscosity, and in part to its adherence to the surface of the peritoneum. In virtue of these same characteristics the intestines and omentum are often agglutinated. This cohesion may be so intimate that forcible separation may threaten the integrity of one or the other of the gut walls.

The material may amount to many liters, distending the abdomen to an extreme degree. It may surround all of the viscera, even filling the space between the liver, spleen, and diaphragm. In this tendency to even distribution it resembles the peritoneal exudates and is unlike any neoplastic process.

The masses and nodules often described are sometimes true metastatic neoplastic processes, but sometimes represent amorphous mucinous masses. McCrea and Coplin describe these nodules in their case as composed of a capsule 1 to 3 mm. in thickness, enclosing a soft gelatinous substance. Running in from this capsule were fine trabecula formed of imperfectly developed fibrous tissue. There was no epithelium, round cells and leucocytes being the sole cell representatives.

The wall of the mother cyst is grayer than that usually found in pseudomucinous cysts and is more easily torn. In this they resemble the serous cystadenomata of the ovaries. In this, perhaps, as in their clinical habit, they lean toward the more malignant types of ovarian cysts.

The microscopic appearance may best be considered from the viewpoint of the theories of the nature of the disease, namely, the changes in the peritoneum itself and the cyst contents.

Virchow was the first to note that there were distinct changes in the peritoneum. These consist in the usual reactions to irritation, fibrinoid degeneration of the connective tissue bundles and, as

Fränkel pointed out, in the disappearance of the elastic fibers. At a distance from the direct contact with the foreign material the peritoneum responds only by an increased vascularity.

Over the masses a fine pellicle of fibrin takes place. That this is a product of the peritoneal exudate can be shown experimentally. It is the organization of this which produces the fine vascu-

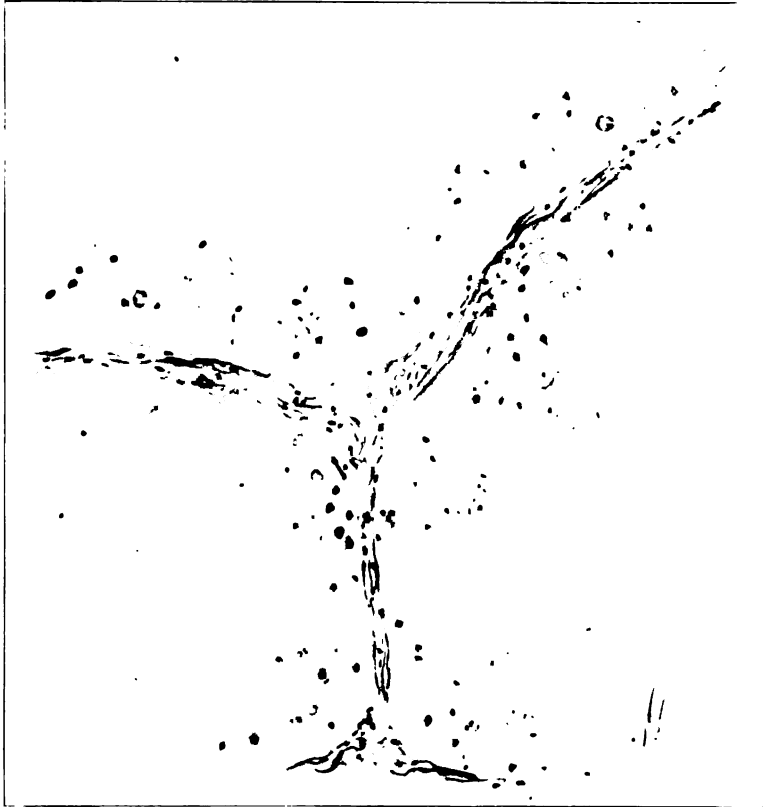


Fig. 230.—Pseudomyxoma of the peritoneum. Alveolar formation is suggested but all cells have undergone degeneration. Note the resemblance to colloid carcinoma in Fig. 226.

lar membrane with which some of the globular masses are covered. Beneath the masses the peritoneum responds by the production of fibrous septa (Fig. 230) which extend between the lobulations of the masses. The presence of colloidal material between these fiber bundles together with stellate cells may be regarded as products

of myxoid degeneration, or as Westphal thinks, may be due to the plugging of lymphatics by colloid masses in the process of absorption. Though this idea has the stamp of Virchow on it, modern tinctorial chemistry makes it difficult to uphold. These account for the difficulty of removing the material from the peritoneal surfaces. These septa may branch like a tree, or the various branches may coalesce, forming cavities in which the material lies. These may be free from cells or may carry on their surface endothelial-like cells and polynuclears near the peritoneal surface.

The colloidal material is homogeneous at most, palely staining but may show laminations, the various layers of which may show variations in coloration. The peritoneal base may show hypertrophy and as in the case of the appendix the reaction may be sufficient to cause attachments to surrounding structures, sufficiently great to wall off the organ in a measure.

In these instances in which a distinct columnar epithelium has been described the basement tissue is edematous and the cells in a state of beginning disintegration (cf. McCrea and Coplin). These changes presage the early destruction of the cell and may account for many areas containing no cells.

Evidence of deeper invasion of the tissue is not lacking. The most marked example of this is recorded by Myer in which cystic processes penetrated the spleen and produced openings into the colon. In Polano's case they followed along the portal vein into the liver. In following along the tract made by the trocar as in the cases of Baumgarten, Sanger and Peiser, one is reminded of the late metastases in papillary cystadenomata.

**Symptoms.**—The symptomatology of the appendiceal and ovarian type is sufficiently divergent to warrant a separate consideration of the two types. The symptoms in neither lead to more than a suspicion of the nature of the disease.

**Ovarian Type.**—Uneasiness amounting to actual pain with abdominal distention are the usual complaints the patient brings to the practitioner. Chills and sweating are mentioned by Smith. Sometimes a history of an acute pain is presented, as in Lewitzky's case, in which two attacks simulating a generalized peritonitis preceded the discovery of the tumor. The cause of these pains can not be determined. That it may herald the rupture of the cyst is

clear. I once operated on a patient stricken with severe abdominal pain. The discovery of an ovarian tumor seemed to warrant the diagnosis of a twisted pedicle. At operation the pedicle was not twisted, but one compartment of a multilocular pseudomucinous cyst had ruptured, producing a marked reaction on the part of the peritoneum. Whether such pains are due to rupture of the cyst or some other accident can not be stated. The distention is generally symmetrical, though sometimes one side presents the greater enlargement. The degree of distention varies greatly. The diaphragm may be pressed upward, making respiration difficult. In one of Atlee's cases this was the symptom that demanded interference. Sometimes the mother cyst can be palpated more or less definitely. Percussion waves across the mass may be elicited. Wendler made the observation that this wave traveled more slowly than in ascites. Myer finds the fluctuation uncertain and sees in the indefiniteness a symptom of value. In some instances, on the other hand, ascites of a simpler character may long precede the development of gelatinous material, as in the case of McCrea and Coplin. It is possible that ascites is always the first response on the part of the peritoneum. In my case in which rupture preceded operation by two weeks, a serous exudate was present.

The percussion note may be dull over the entire abdomen, but usually there is tympany in some region, usually asymmetrically distributed. There is always tympany in the epigastrium.

Vaginal examination gives evidence of a varying character. In some cases the uterus is high, barely palpable, as in Myer's case, or it may be deep in the pelvis and massed in by colloidal material, or any degree of variation between these extremes may exist. The high location of the uterus is often noted.

*Appendiceal Type.*—In many of the appendix cases one or more attacks of acute pain precede the discovery of the disease. Since a proximal occlusion is essential, an inflammatory attack likely always precedes, save in one case in which a carcinoma caused the occlusion, namely, that reported by Hüter. Sometimes the discovery of the gelatinous mass is brought about because of the sudden onset of severe pain as in Morris's case. Goldschwend had a similar case. Neumann's case had had two typical attacks of peritonitis as did also that of Chavannez.

The repeated attacks of pain may in some instances be due to successive ruptures of the appendix with escape of its mucinous contents. Cysts remaining after the appendix has been removed then present the same symptoms as the rupturing appendicitis. MacLean observed one case in which the intermittent discharge took place twelve times.

Aside from the history of previous pain the discovery of an indefinite tumor is about all that has been recorded that might aid in the diagnosis.

**Diagnosis.**—A positive clinical diagnosis has been made only when the trocar was employed. Wendler succeeded in securing a few drops of the material through a large trocar, as did Atlee. Spiegelberg demonstrated the danger of this manner of obtaining evidence in that a fatal peritonitis followed one of his attempts. Negative results following puncture are of importance. Exploratory incision is safer. Meyer believes that a differentiation should be made, and presents a table designed to aid in differentiating this condition from ovarian cyst. The abdominal disturbance and rate of growth, according to him, are greater than in the case of ovarian cysts and fluctuation is less pronounced. In a cyst with a twisted pedicle, on the other hand, the pain may be greater, and there may also be a more pronounced febrile reaction and a rapid increase in size. In pseudomyxoma from the appendix there is greater difficulty in demonstrating the tumor by palpation by vaginal examination than in the case of cyst origin.

In contrast to retroperitoneal tumors edema is rare. In only one case, Geyl's, was edema of the feet a marked symptom.

The previous removal of an ovarian cyst should always excite suspicion when a renewed abdominal enlargement begins. Many cases recorded bear this out. Polano had a patient with a free interval of more than two years. Eden also had a patient who showed a renewal of the disease in the other ovary after two years of freedom.

The type developing from the appendix can be suspected only in cases in which there have been previous attacks of appendicitis and they present an indefinite mass in the region of the appendix.

After the available symptoms have been employed for the purpose of forming a diagnosis, curiosity will excite the average

individual into an exploratory incision. Examination for ovarian tumor, or cystic disease of the appendix reveals the site of origin. In McCrea and Coplin's case incision did not reveal the source of the disease.

**Prognosis.**—In some instances the removal of the primary offender seems to check the disease. This is particularly true of the appendiceal type. In ruptured cysts the chances for recovery are less, though it is possible even after considerable extension on the peritoneum. We recognize here again an analogy to the papillary cystadenomata. When the disease has once become fully established, recurrent development of the material takes place with an invariably fatal result. This may be deferred even to a number of years, however.

Statistics avail but little in arriving at a conclusion, for late recurrence is common. Günzberger in 44 cases collected from the literature found 17 recoveries. Bettmann reports 18 recoveries in 35 cases. Schurmann found 39 recoveries and 16 deaths. As is usual with statistics in malignant disease the results sound better than is borne out by clinical experience. Schurmann after stating the figures above quoted begins the next paragraph with the statement "Recurrence is almost universal." Sometimes secondary operations achieve results. Pfannenstiel quotes a case in Fritsch's experience in which the patient remained well eleven years. Gottschalk reports one still well after four and a half years.

Death takes place in a surprisingly large number of cases because of sepsis or embolism. This was true in eight out of eighteen cases reported by Honecker.

**Treatment.**—The treatment consists in removing the offending lesion and the removal of as much of the gelatinous material as possible. The appendix usually is easily removed and the remains of ovarian cysts are usually removed by simple ligation of the pedicle. It was formerly the practice to remove the colloidal material by irrigation, but the more recent practice has been to remove as much as possible by manual means. The former practice of placing a drain has also most properly been given up.

It is a question whether actinic rays exert a favorable influence or not. In a recent case in which metastatic colloidal tumors of the peritoneum appeared, temporary improvement was coincident with the use of x-rays.



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